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BENIGN TUMORS OF THE ESOPHAGUS

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Hines, Ill.

Benign tumors of the esophagus are rare lesions. There have been an increasing number of reports of these lesions within the last few years. One report states that there were about 200 of these cases recorded up to 1950. This is a report of 12 benign tumors of the esophagus found at the Hines Veterans Administration Hospital.

DISCUSSION OF CASES

A history of two polyps was found in the records of the hospital. One was discovered at autopsy and, as far as could be determined, it presented no symptoms during life. The other case was discovered in 1938. The patient presented difficulty with swallowing and complained of a lump in his throat. He complained that swallowing was more difficult when something cold was swallowed. Esophagoscopy showed a polypoid mass 32 cm. from the incisor margin. A benign lesion was found on biopsy examination. No treatment was given and no further follow-up has been obtained.

The next case report is that of a Negro man, 40 years of age, admitted to the hospital in May 1953, vomiting of large quantities bright red blood, and passing large tarry stools. His condition was desperate and a total of 12 units of blood were given in a very short time. Exploration of the abdomen was deemed an emergency, but the bleeding apparently stopped and operation was deferred. Continued questioning elicited the complaint of vague epigastric distress of two months' duration, which was relieved by antacids. This distress was intermittent. A roentgenologic study showed a normal stomach and duodenum with no evi-

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dence of varices. During fluoroscopy of the esophagus, a deformity of the upper esophagus was noted and the esophagus seemed to be displaced backward and the trachea was displaced forward. A lateral roentgenogram of the esophagus showed some widening in the retrotracheal area. Figure 1 showed some barium in the cervical esophagus which was pushed away from the trachea by a mass appearing to be about 2.5 by 5 cm. in size. The trachea was clearly visible and it seemed to be pushed forward slightly. Figure 2 shows an anteroposterior roentgenographic view of the same area which shows that the esophagus has been flattened out by a mass so that it is much wider than normal, and seems to be compressed. The roentgenologic diagnosis was an enlarged thyroid gland occupying the space between the esophagus and trachea. On esophagoscopic

FIG. 1. Lateral view of barium swallow showing space-occupying lesion between trachea and esophagus.

examination, a submucosal mass was suspected just below the cricopharyngeus muscle, not occluding the lumen. This mass could be compressed by the esophagoscope, but would resume its shape on withdrawl of the instrument. Tissue for biopsy was taken. Fortunately, a portion of the tumor was not obtained and the pathologic report was normal mucosa. A benign tumor of the esophagus was considered—most likely leiomyoma—but the thyroid enlargement was considered more likely.

Exploration of the neck was made in July 1953. A collar incision, with division of the right strap muscles, was done. The right thyroid lobe was mobilized and elevated so that the space between the trachea and the esophagus could be explored easily. A mass occupied this space which was about 2.5 by 5 cm. in size. This mass could not be freed from the esophagus, but could be freed partially from the trachea. It could not be enucleated, and was removed partially by morcellation. Bleeding was profuse so the method of excision was clamping, excising, suturing and then repeating the process until most of the tumor had

been removed. We believed this was an hemangioma and microscopic study showed multiple angiomatous spaces. A diagnosis of cavernous hemangioma was made. The patient's recovery was uneventful and now he is doing full time work; feeling fine, with no further bleeding or distress. Repeat roentgenograms show a marked narrowing of the space between the trachea and the esophagus. Operation in this case produced some scarring. A diagnosis was made and bleeding was controlled, but the tumor could not be removed completely.

This case is an example of massive gastrointestinal hemorrhage due to a cavernous hemangioma in the esophagus which ruptured, possibly as a result of some foreign body eroding the surface.

FIG. 2. Anteroposterior view showing flattening and distortion of the esophagus with compression.

Eight leiomyomas of the esophagus have been found at the Hines Veterans Administration Hospital since 1946. Four were treated surgically and four were discovered at autopsy. One was discovered during operation when a vagotomy and gastroenterostomy was being done for peptic ulcer. The leiomyoma was at the lower end of the esophagus. It was enucleated during the exposure of the lower esophagus for transabdominal vagotomy. None of the 8 patients presented symptoms that seemed to involve the esophagus. One patient had minimal epigastric burning, and on roentgenologic examination a shadow was seen, which seemed to be an extra-esophageal mass. Figure 3 shows the lesion that was removed at operation. Because it almost encircled the esophagus, in a grip-like manner, it was thought that it could not be removed by enucleation. An esophageal resection was done with esophagogastrostomy. Recovery was uneventful. A similar specimen was removed at autopsy from another patient who died of

FIG. 3. Surgical specimen showing leiomyoma almost encircling the esophagus

unrelated causes. Detailed questioning of the relatives revealed no symptoms at any time.

The preferable treatment, if possible, is to enucleate the tumor, and in the following case, this was possible. This man had some vague symptoms of epigastric distress; was seen by his private physician who ordered a roentgenogram. An abnormal shadow was seen and he was referred for further study. On barium swallow, there was a defect in the esophagus which was smooth, and the presumptive diagnosis was leiomyoma (fig. 4). Thoracotomy was done and a leiomyoma was enucleated easily. Figure 5 shows the specimen. The postoperative film is shown in figure 6. Recovery was uneventful and he has had no symptoms

FIG. 4. Oblique view of barium swallow showing mass compressing the midesophagus

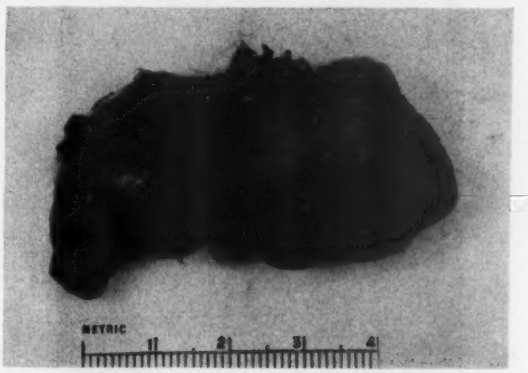

FIG. 5. Enucleated surgical specimen from case shown in figure 4

since that time. The enucleation was done by incising the longitudinal muscle and enucleating the tumor without opening the esophageal mucosa.

That these tumors, even though they are not causing symptoms, should be removed, hardly deserves discussion. The following case is presented to illustrate what may happen in some of these cases. This 67 year old white man complained of painful swallowing of four months' duration. Barium swallow (fig. 7) showed

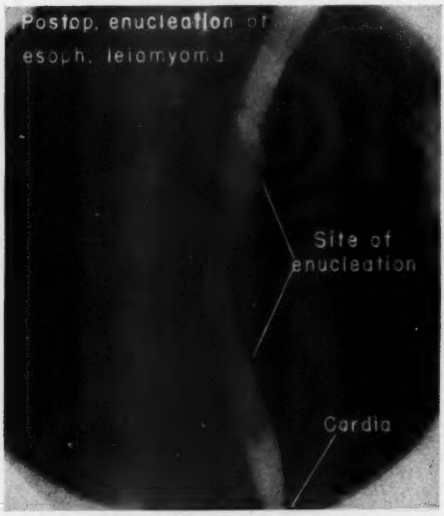

FIG. 6. Postoperative view of case shown in figures 4 and 5

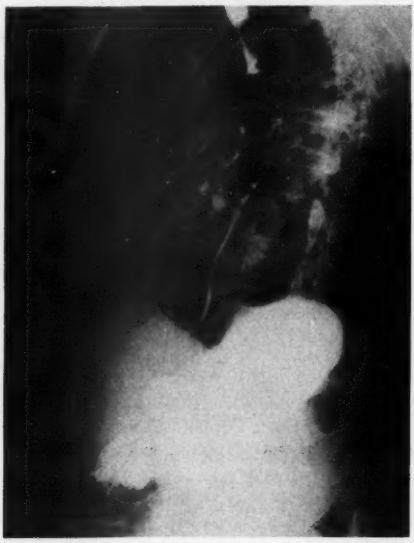

FIG. 7. Oblique view of barium swallow showing irregular filling defect

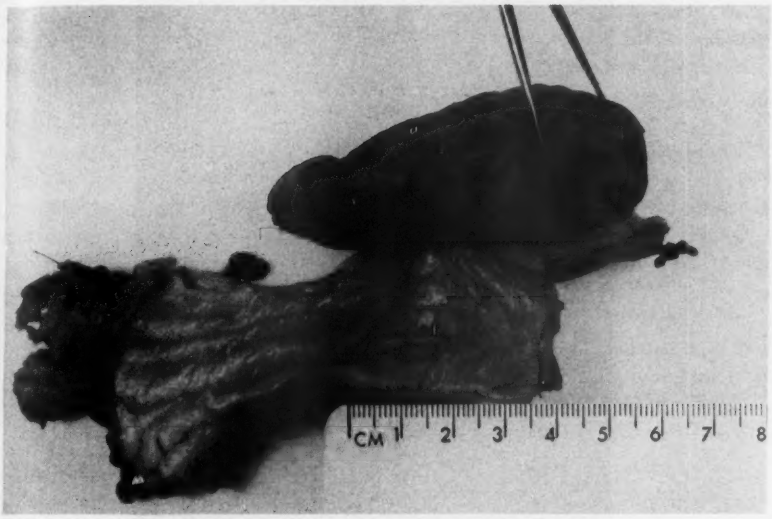

FIG. 8. Surgical specimen of patient shown in figure 7 showing leiomyosarcoma of esophageal surface.

a deformity, and on esophagoscopy, a fungating lesion was seen. A biopsy was interpreted, on frozen section, as a squamous cell carcinoma, very anaplastic. Resection therefore was done. The specimen shown in figure 8, is certainly an angry looking lesion protruding into the esophagus. The esophagus was resected well above the tumor. It must be remembered that, after removal, the tumor did not retract, but the esophagus did retract so that it may appear on the specimen photograph that the removal incision was closely adjacent to the tumor. A follow-up roentgenogram, taken some weeks after operation, showed the gastroesophageal anastomosis was functioning well. This patient has been comfortable and in good condition for the past one and one-half years. On later pathologic examination the tumor, previously called a carcinoma, was found to be a leiomyosarcoma. We believe that it is reasonable to assume that this man had a leiomyoma which caused no symptoms, but later it became sarcomatous, causing symptoms.

The next case is of interest in that it presents another example of both benign and malignant tumors in the same patient. This patient, aged 42, a laborer, admitted drinking two to three pints of whisky daily for many years. He complained of substernal chest pains with radiation of this pain to the right and left chest. Loss of weight was 21 lbs. A roentgenologic study of barium swallow was interpreted as negative. Esophagoscopy was done and a suspicious lesion was sectioned for biopsy. Squamous cell carcinoma was found. On rechecking the roentgenograms a suspicious area was found (fig. 9). It requires a certain degree of the use of Rigler's *retrospectoscope* to be sure that there is anything significant. Esophageal resection and esophagogastrostomy were done. Exami-

FIG. 9. Oblique view of barium swallow showing irregularity at site of benign and malignant tumor.

FIG. 10. Low power showing (a) carcinoma of the esophagus and (b) benign intramural tumor 4 mm. apart.

nation of the specimen (fig. 10) shows an unusual arrangement. At a. there clearly is a squamous cell carcinoma, and at b. there is a benign tumor which had been called leiomyoma by two pathologists, a benign granular cell myoblastoma by one, and a neurofibroma by another. We believe this is a leiomyoma, but at any rate, it is benign.

The close proximity of a benign and malignant tumor in the same patient was at the time believed to be a mere coincidence. However, Daniel and Williams[6] have reported a similar condition in which a leiomyoma was complicated by islets of carcinoma. Callanan[3] recorded the simultaneous occurrence of a benign and malignant tumor in the esophagus and stated "Could the benign tumor have delayed the passage, and the simple tumor indirectly aided the development of the malignant one?"

Our case is the third one we can find in which this coincidence has occurred. We merely can suggest that a benign tumor may invite later malignancy in the esophagus.

SUMMARY

Twelve cases of benign esophageal tumors have been reported.

A cavernous hemangioma presented an unusual diagnostic and therapeutic problem.

Leiomyomas should be treated by surgical excision—either resection or enucleation.

Malignancy in the esophagus may be invited by benign tumors. Two cases are reported that suggest some relationship between benign and malignant tumors.

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NITROGEN METABOLISM IN THE SURGICAL PATIENT

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Surgical trauma initiates a multitude of chemical and physiologic changes in the body which are recognized readily during the course of an operative procedure and in the immediate few days following an operation. During convalescence these changes are reversed in ways still obscure to the investigator. Convalescence ultimately leads to a physiologic state comparable to the pretraumatic or preoperative one. The relationship between surgical trauma and the physiologic and metabolic alterations observed in the post-operative state is complicated. The few facts known have been gleaned from arduous labors by many investigators.[1-19] The difficult task of sorting out the many metabolic interrelationships existing in surgical convalescence should encourage everyone to exhibit restraint in voicing categoric opinions.

During the past two and one-half years 168 adult patients have been studied on the metabolic division of this hospital. The observations made upon these patients have afforded us an excellent opportunity to study the physiologic responses of the patient to surgical trauma. Definitive answers, however, to the many problems existing in the field of investigation will come from the laboratories, and in many instances only after new technics of investigation have been devised.

Nitrogen metabolism is a small but vital part of all the metabolic activity going on in the patient. It now is generally recognized that the proteins and amino acids of the body are in a constant state of dynamic equilibrium. Anabolic and catabolic reactions go on constantly and simultaneously. Shortly after amino acids have been administered either orally or parenterally they cannot be distinguished from amino acids that were in either the blood, liver, or muscle prior to the administration. When a nitrogen deficit exists it frequently is difficult to know whether there is an impairment of anabolic activity or an augmentation of catabolic reactions. Little is understood of the specific mechanisms that alter the nitrogen equilibrium of the body one way or the other. It is important, however, from the conceptual standpoint to appreciate that amino acids in the plasma, lymph, and tissues are indistinguishable from administered amino acids.

Surgical trauma produces alterations in nitrogen metabolism and certain broad patterns can be described, although there is considerable individual variation from one patient to another. A knowledge of these patterns is of assistance not only in understanding the biologic response of the body to trauma, but in managing the progressive recovery of seriously ill surgical patients.

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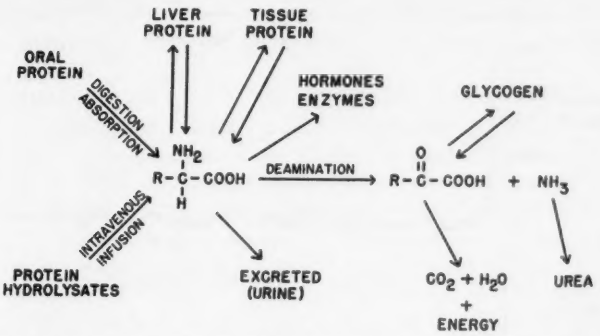

FIG. 1. The metabolic fate of circulating amino acids is shown. Some of the reactions are reversible. When calories are insufficient to provide for minimum energy requirements, the equilibrium between amino acids and the body proteins which always are in a state of dynamic change is altered and more amino acids are deaminated permitting the release of energy.

The pattern of nitrogen response to trauma is influenced or modified by many factors among which are age, sex, part of the body traumatized, renal function, electrolyte balance, fluid administration, complications of surgery, hormonal changes, and the energy requirements of the body. In addition to these, there are four aspects of nitrogen metabolism that are important for the surgeon to comprehend. First, is the catabolic response to trauma; second, the influence of pre-existing protein depletion upon the nitrogen metabolism of a patient undergoing a major surgical procedure; third, the effect of a previous operation or injury upon nitrogen metabolism when a second procedure is done within two to three weeks of the first; and fourth, the relationship of nitrogen metabolism to the total caloric intake of the patient.

It has been known since the early part of the century that a normal adult will degrade or break down considerable quantities of body protein when he has contracted a serious infection such as typhoid fever,[18] or has been exposed to either accidental or surgical trauma. This information was obtained by measurements of the total quantities of nitrogen entering and leaving the body. A loss of more nitrogen than the intake is circumstantial evidence that body protein is being hydrolyzed to amino acids which then are deaminated and oxidized with administered amino acids finally to form carbon dioxide, water, and urea (fig. 1).

The extent and rate of destruction of tissue protein, as well as the inability of the body to utilize administered amino acids for protein synthesis, has been considered by most investigators to parallel the severity of trauma, although no studies have been made in which the severity of the trauma could be more than grossly estimated. The patient subjected to an inguinal herniorrhaphy done with a minimum of trauma may display little if any nitrogen wasting. If it does occur, a normal nitrogen balance is restored within several days following the operation. On the other hand a patient subjected to an extensive traumatic procedure such as a gastrectomy, splenectomy, and partial pancreatectomy may

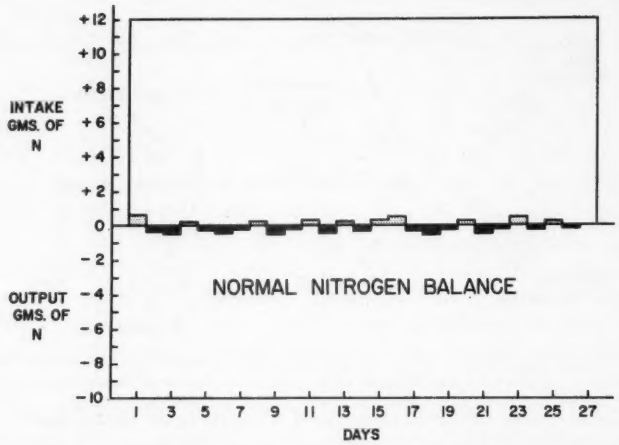

FIG. 2. This chart and all subsequent ones are schematic representations of patterns of nitrogen metabolism. The abscissa designates days before and after operation where an operation is written vertically above the center line. The ordinate consists of nitrogen intake in grams above the center line. Nitrogen output is represented downward from the top of the line where nitrogen was provided, or downward from the center line where no intake of nitrogen was provided. A positive balance is demonstrated by the stippled area above the center line and a negative balance by the black area below.

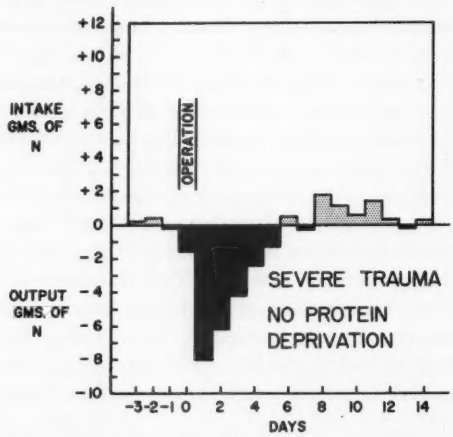

FIG. 3. This demonstrates the obligatory negative balance occurring in the presence of an adequate protein and caloric intake to provide for minimum requirements and continued throughout the hospital course. This is the type of response seen in a healthy adult subjected to a complicated, prolonged abdominal or thoracic procedure.

show evidence of excessive protein degradation for many days. Patients do not recover from the effects of an operation unless this catabolic process diminishes and the body accepts nitrogen for the restoration of lost protein. Figure 2 shows schematically a normal nitrogen balance in a healthy adult. Figure 3 shows the marked loss of body nitrogen following operation in spite of the continuous

administration of 12 Gm. of nitrogen per day. The excessive catabolic activity ceased spontaneously, however, as it does in all patients who recover from operation.

Why the healthy adult patient following trauma goes through a period when he destroys protein and exhibits a reduced capacity to utilize administered amino acids for tissue synthesis is not known, although numerous investigators have attempted to determine the cause. No one has been able to determine scientifically whether this nitrogen wastage is beneficial or injurious. Since it is known that protein in these circumstances is used largely for energy purposes, it is very likely that the energy requirements (which are not known) of the immediately postoperative patient initiate the hydrolysis of body protein.

When a healthy patient suddenly is deprived of all protein or his diet is reduced drastically, a characteristic change in the pattern of his nitrogen metabolism occurs. This is represented in figure 4. Large quantities of nitrogen are lost initially from the body, primarily in the form of urea through the kidneys. Within a short period of time this large quantity of excreted nitrogen diminishes and from 2 to 4 Gm. (providing he has a good caloric intake) are lost in each 24 hour period. What this small quantity of excreted nitrogen represents is not understood clearly but has been presumed to be the result of the minimal obligative deamination that goes on continuously. When the patient who has been depleted of protein for many weeks again is given an adequate protein intake in the form of either protein hydrolysates administered intravenously or oral protein a strongly positive nitrogen balance results for several days until a normal balance finally is attained (fig. 5). When a patient who has been deprived chronically of protein is subjected to a major surgical procedure, the pattern of nitrogen balance (fig. 6) is considerably altered from the characteristic response observed in the healthy patient. Instead of finding a marked increase in the quantity of nitrogen excreted or an exaggeration of the negative

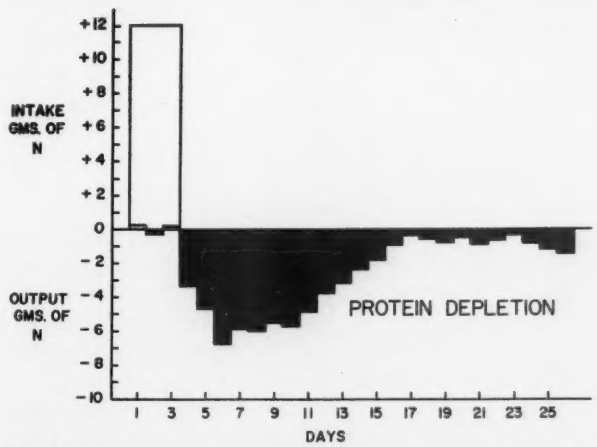

FIG. 4. When protein intake ceases, the initial loss of nitrogen is large, becomes progressively smaller, but never disappears.

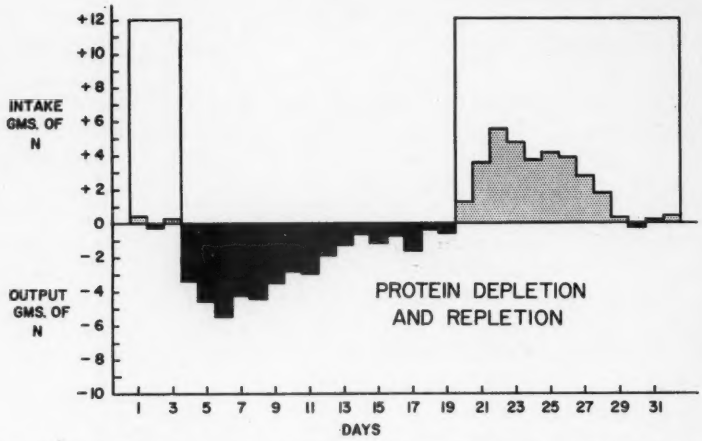

FIG. 5. The institution of a strongly positive nitrogen balance is striking when the protein depleted patient is given sufficient protein and calories either orally or intravenously.

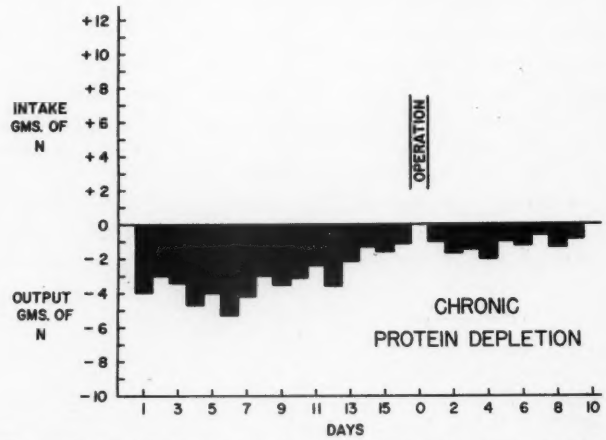

FIG. 6. The marked catabolic response to surgery ordinarily seen in the healthy adult is lacking when chronic protein depletion exists.

balance, there is no more than a slight transient increase in the quantity of nitrogen excreted. The intensity of the biologic response is reduced markedly. The body jealously preserves its residual protein when it cannot afford to be extravagant. The homeostatic properties of protoplasm frequently permit the display of more intelligent behavior than do many forms of adult cerebration. This in no sense implies that a patient depleted of protein is a better surgical risk or that his convalescence is going to be smoother. Neither is true.

The patient who has two major surgical procedures within a short period of

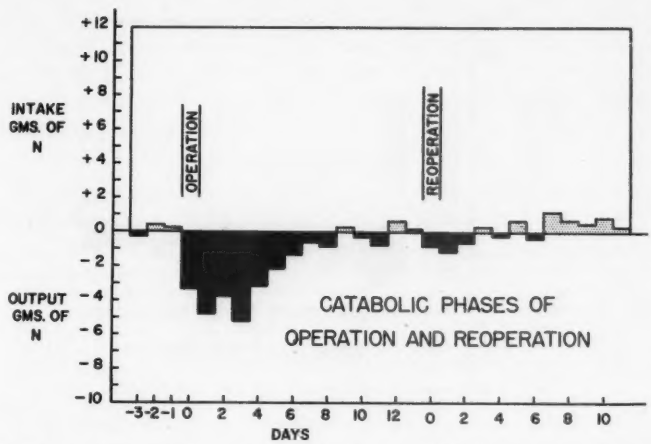

FIG. 7. The catabolic response to reoperation in a patient having been provided with sufficient protein and calories is negligible when compared to the response following the initial procedure.

time frequently manifests a much less intense catabolic response to the second operation. It has been thought by some that the initial operation in some way conditioned the patient to a diminished secondary response. An examination of the records in this laboratory and published protocols from the work of others suggest that a diminished secondary catabolic response to surgery occurs in the presence of either acute or chronic protein depletion. The patient actually may be in an anabolic state indicated by a positive nitrogen balance at the time of the second operation, but this in itself does not indicate that all previously depleted protein has been restored. Figure 7 shows a secondary response when the patient is just beginning to attain a positive nitrogen balance.

The relationship of the total caloric intake to the degree and extent of the catabolic nitrogen response to trauma has been a controversial subject for many years and definitive answers are not existent at the present time. One factual observation has been made in many laboratories including our own; i.e., that the quantity of nitrogen lost from the body during the first few days after a surgical operation can be reduced by providing the patient with a large number of calories in the form of nutrients that are oxidized readily for energy purposes. Most observers believe that the length of time that a patient remains in negative nitrogen balance can be influenced favorably by a caloric and protein intake above the minimum required. This obviously does not imply that if a patient is given 75 to 100 Gm. of protein and 3500 calories a day he will not go into positive balance at an early date. He will, but only because the protein is available to the cells of the body at the time when they are prepared to accept the nitrogen provided for the synthesis of new protein. By giving more protein and calories the return to positive balance cannot be hastened. On the other hand, by providing a protein intake of 75 Gm. and reducing the total calories to 800

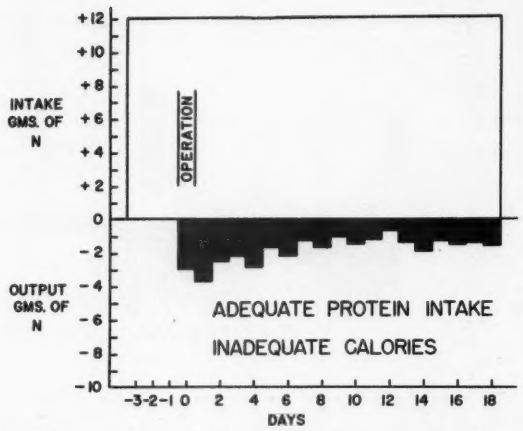

FIG. 8. This chart demonstrates a continued negative nitrogen balance although 12 Gm. of nitrogen are being supplied daily. The deficit is in the total number of calories provided which places demands upon the body to provide its own energy, partly through the destruction of its own protein.

(fig. 8) far below minimum requirements, the onset of the anabolic part of convalescence may be delayed many days or even weeks.

One of the many bits of information that would be of considerable assistance in understanding the biologic response to trauma is the energy requirements of the postoperative or injured patient. Only indirect evidence is available at the present time to permit any estimation of the total number of calories required by a healthy adult who has had a combined abdominoperineal resection 24 hours previously. It is estimated that from 2500 to 4000 calories are required for such a patient, but exact studies are lacking.

The manner in which intravenous hexose and amino acid preparations are administered may make a certain difference in how the amino acids are utilized.[9, 13] If hexose solutions are given just prior to the administration of amino acids, some of the immediate energy requirements of the cells are met and the amino acid nitrogen is more available for the restitution of tissue protein. If the order is reversed, the amino acids are deaminated rapidly and oxidized for energy purposes. There is some evidence to bear out this hypothesis and it is quite possible that a significant difference in nitrogen retention might be obtained by the appropriate administration of hexose and amino acid preparations.

CONCLUSIONS

The metabolic disturbances that occur in most surgical patients are mild and temporary. Convalescence is progressive and rapid. When nutritional and electrolyte disturbances exist prior to operation, when the magnitude of operative trauma is great, and when convalescence is impeded by a series of complications, considerate attention to the disturbed metabolic processes can mean the differ-

ence between death and ultimate recovery of the patient. Fortunately the homeostatic capacities of a human being's sick protoplasm are beyond imagination. A knowledge of some of the factors that influence metabolic reactions in the surgical patient permit the surgeon to assist, in a purposeful fashion, the convalescence of his patients.

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OVARIAN CANCER—PROPHYLACTIC OOPHORECTOMY

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All ovarian enlargements potentially are dangerous. This is a well known fact. Most tumors of the ovary, benign or malignant, are asymptomatic and the malignant ones usually enlarge and extend beyond the ovary without producing signs or symptoms of their existence. Many of these have extended to the *point of no return* before they have been discovered. We have all seen patients with extensive tumors of relatively long standing but whose complaints are of a short period. The patient hardly notices the minor disturbances caused by the advancing cancer until, perhaps, on careful questioning, she recalls some slight enlargement of the abdomen for the past year or so, or that she had some urinary symptoms or minor intestinal complaint, and then upon opening the abdomen an inoperable, incurable ovarian malignancy is found. This form of creeping death which too often progresses without warning to this stage of incurability, this *silent menace*, as H. S. Crossen[3] termed it, has baffled gynecologic surgeons up to and including this moment. These facts, considered together with the poor prognosis of this disease when it is not discovered early, have led our teachers to the conclusion that, because periodic health examinations on a large scale seemed impractical, the most expedient method of decreasing the number of deaths from ovarian cancer is to remove the ovaries before the cancer develops. Randall,[4] being one of the first to challenge this line of thought, has said, "As yet we have learned of no treatment which justifies denial of so practical a solution of the problem—if the incidence of ovarian carcinoma warrants it."

In a review of 200 ovarian tumors seen on the gynecologic service in The Ohio State University Hospital, Spencer and Reel[6] found an incidence of 0.4 per cent malignancies in women over 50 years of age. None of these women had had previous pelvic surgery during their fifth decade at which time prophylactic removal of the ovaries could have been carried out. From the files of our Gynecologic Tumor Conference at The Ohio State University Hospital we can report an additional 105 cases of ovarian malignancy, none of which fall into this class of patients who had pelvic surgery between the ages of 40 and 50 years where prophylactic removal of the ovaries could have been done. These obviously are small groups and probably not of statistical significance, but the findings agree with the published reports of Speert,[5] and Clark and Judge,[1] who also found that the vast majority of women eventually developing ovarian carcinoma had not been subjected previously to pelvic laparotomy during which time so-called prophylactic oophorectomy could have been done. I would like to suggest, although I have no way to prove it, that a very much higher percentage of these

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women had probably not had a pelvic examination within—not six months, not one year—but five years prior to the discovery of their ovarian malignancy. In a further effort to discover the true incidence of ovarian carcinoma, let us again turn to Randall who quotes The Cancer Morbidity Reports of New York State for 1949 to 1951 in which there is given a table of a woman's probability of developing ovarian cancer. The risk is calculated at about 0.8 per cent for age 50 years. In other words, a woman of 50 years has 0.8 per cent chance of developing an ovarian cancer before she dies. If 20 per cent of these women are cured, then approximately 640 women per 100,000 female population over 50 years of age would die of ovarian cancer. This is not the case, and the risk of developing ovarian cancer at age 50 years is not just slightly less than 1 per cent: it is much less than that.

From the vital statistics records of the State of Ohio for 1952 and 1953 (which give an incidence of approximately 9.1 deaths from ovarian cancer in all age groups per 100,000 female population), and the State of Connecticut[2] for 1943 (giving an incidence of deaths from ovarian malignancy at all ages of approximately 6 per 100,000), the deaths from ovarian cancer per 100,000 female population is less than 10. The incidence rate of new cases of ovarian cancer for New York State for the years 1942 to 1944 was approximately 25 per 100,000 population (of all ages). In figures released by The Metropolitan Life Insurance Company the number of new cases of cancer of the ovary, in selected urban areas in the United States (1947 to 1948) for all ages is 14.7 per 100,000 female population and averages 37.3 per 100,000 female population for women over 50 years of age. Furthermore, the incidence of death from ovarian (fallopian tube and broad ligament) cancer in the whole United States in 1950 was approximately 8 per 100,000 female population. All of these figures suggest a true incidence of ovarian carcinoma of less than 40 per 100,000 women over 50 years of age, not 640 as previously mentioned. This is enough, and no matter from which side you look, cancer of the ovary has an unfavorable over-all picture, but this primarily is due to the fact that it is so badly neglected. As you know, in a small favorable group 80 per cent are cured, but the gross cure rate is about 20 per cent. Only the solid adenocarcinoma, which is completely undifferentiated, has a hopeless outlook and it is seldom discovered at an early stage. Fortunately it is rare.

The incidence of ovarian carcinoma and the incidence of death from this disease certainly is a difficult one to calculate accurately. This results from several factors which cannot be too well controlled. Death certificates may indicate ovarian carcinoma as the cause in instances of carcinomatosis or ascites when no operation or autopsy has been done to verify the diagnosis. Vital statistics records of most states and the United States include cancer of the fallopian tubes and broad ligaments with those of ovarian cancer. On the other side of the ledger, patients who have a primary or recurrent ovarian carcinoma which shortly would have been the cause of their death may die of an intercurrent disease. In addition to this, statistics taken from selected urban areas, particularly those incorporating large medical centers, may be inaccurate because of

out of town patients registering from local adresses of friends or relatives. These factors, however, probably tend to cancel each other with a result that the statistics we have at hand are reasonable.

From the references which I have quoted, (2, 4, 5, 6, 7), it seems that the highest incidence of ovarian cancer is approximately 37.3 per 100,000 female population in women over 50 years of age. This, as would be expected, is slightly higher than it is for the 45 to 49 age group (31.7 per 100,000). In considering the practicability of prophylactic oophorectomy this incidence obviously would be reduced by patients having hysterectomies for malignant conditions of the uterus and ovary, and by those in whom there is indication for bilateral oophorectomy for benign conditions of the ovary itself. In weighing all of these facts it seems that the best we could accomplish would be the prevention of subsequent ovarian cancer in 3 women out of every 10,000 hysterectomies. In contact with these 10,000 women the surgeon would have a most excellent opportunity to stress the importance of subsequent pelvic examinations. Another point to consider, although it, too, has nothing to do with the prevention of ovarian cancer, but is a very important matter in stressing the urgency of periodic pelvic examinations, is the fact that the majority group of ovarian cancers has a better prognosis than the minority group. When we remember our poor results this seems paradoxical. Although, specifically, when we consider the over-all salvage, the cystadenocarcinomas constitute by far the largest group of ovarian malignancies, but have, in general, the best five year survival rate. The probable reason for this is that the cystadenomata pass through a relatively long stage of complete resectability. That is, there is a period in their development that is long enough that they may be discovered and excised before metastasis occurs. This is and has been true for many years. It seems then that by further effort on the part of all physicians this salvage could be very much increased by forcing many more of these tumors into the favorable group of the 80 per cent cure rate. If we consider this as a distinct possibility, plus the number of women who will never develop ovarian carcinoma, it does not logically follow that so-called prophylactic oophorectomy can be of much real value.

At the present, no matter what type of tumor or in what organ it occurs, early diagnosis is the most important phase of the entire cancer problem. It most particularly is true of ovarian cancer which, as we have said, has a gross mortality rate of 80 per cent. If objections have been raised because of the special attention which has been given to the diagnosis of cancer, it has been on the grounds that the means used, for the most part, are those used in the diagnosis of any disease. This generally is true except for the most important factor—time. A delay of a few weeks in the diagnosis of cancer may be fatal.

A delayed diagnosis almost always is due to some form of gross neglect. It usually has been neglect by the patient. In the Philadelphia study it was shown that the most important cause for patient delay was evasion by one or another psychologic device based on fear of the diagnosis of possible or probable cancer which was to the patient a sentence of death. All of this is bad enough, but worse is that for which the medical profession is responsible. In many instances this

has been by general acceptance of what has been called normal procedure, such as the omission of the pelvic examination as part of a general physical examination. How many, many times have we all been called in consultation to see a hospital patient and have found in the chart: "Pelvic examination deferred"? It has been deferred by the intern, by the resident and by the attending staff, and if it were not for the fact that the patient had a complaint referable to her pelvis she would have been discharged without a pelvic examination. Let me quote from the rules of one hospital: "No patient will be anesthetized preparatory to pelvic surgery unless a record of the pelvic examination is written in the patient's chart." This is all very fine, but it has not become part of the rules and regulations of that hospital primarily to benefit the female patient; it is obviously for the benefit of the field representative of the Joint Commission on Hospital Accreditation. If the rules and regulations of the hospital stated, until her hospital record contained a completed postscript to the usual notation, "Pelvic examination deferred", that "no female patient will be served her diet tray", I believe it would be much more effective. This situation in hospitals represents only a very small percentage of neglect on the part of physicians. It is multiplied many, many times in their offices. It is well understood that all of this problem is one which involves public knowledge and attitudes. It involves policy in medical colleges, clinics and hospitals; thereby, their attending physicians, their students and their patients.

Even though there seems little reason to hope that present methods of treatment can be employed in a manner sufficiently effective to improve the curability, enough facts in regard to principles and technics now are known to increase the ovarian cancer cure rates by more than 200 per cent if they are applied. If the patient is not examined, if the tumor is not discovered, it is as if this knowledge were absolutely nonexistent.

The adage, whom I cannot credit with its first utterance, but which has many times since been spoken, "Ovarian function is altered but does not cease with the menopause", should again be emphasized in this discussion of the advisability of removing normal ovaries to prevent subsequent cancer. The advisability of preserving the ovaries before the menopause cannot be disputed. The severity of the menopausal symptoms following castration is common knowledge. If we have accepted the age of 42 or over, as advocated by Crossen, as the time when prophylactic oophorectomy should be practiced, we have not considered the importance of normal sex steroid metabolism for the vast majority of these women.

From a study of the patients in our cancer clinic the average age of menopause is 48.15 years. In addition to this fact there is indisputable clinical evidence that the ovary continues to produce estrogen years after this average age of menopause. In the study of routine cytologic smears it is found that an astounding number of postmenopausal women who have not had any form of steroid therapy show no evidence of estrogen lack. This fact has been long suspected from knowledge of the histories of menopause as related by patient after patient in our practices. Extra-ovarian sources of estrogen have been demonstrated. The

common experience, however, of gynecologic surgeons in having their patients complain bitterly of a severe menopausal syndrome when ovaries have been removed years after cessation of menses, has established the fact of ovarian estrogenic function long after the menopause. Therefore, in considering this phase of the problem, we must remember that the climacteric is a gradual change and that the actual menopause which occurs at an average age of 48 years is but an outward sign of what has been going on and what will continue to evolve during the next several years of the patient's life.

If 3 women might be saved the ravages of ovarian cancer by the removal of the ovaries of 10,000 patients, and if 1 of these 3 might possibly be cured of the disease, then each of us in a lifetime, by practicing so-called prophylactic oophorectomy, could possibly save one death from ovarian cancer. At the same time we would be plunging 5000 women, or nearly that many, into a surgical menopause. Five thousand hysterectomies, by the way, is quite a project. Remember that these are for benign conditions of the uterus and in women of near menopause age. If a surgeon could do two such procedures per working day it would take him 10 years to accomplish this feat. This, as we have indicated, would represent a good number of hot flushes and headaches, but it would be a boon to the manufacturers of estrogen. I cannot subscribe to all of this.

Further, I believe if every opportunity to examine the patient is utilized and if every opportunity to spread the gospel among women of the importance of these examinations, which must be done often enough to detect this insidious malignancy, infinitely more women can be saved from ovarian cancer than by prophylactic oophorectomy.

I am not sure of the author, but in closing I would like to quote him: "The older my wife becomes, the more I respect the ovaries of other women".

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CANCER OF THE GALLBLADDER: A REPORT OF TWELVE CASES

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Since de Stoll first reported a case of primary carcinoma of the gallbladder in 1772, numerous articles regarding this highly malignant tumor have been written. All of them have shown that the diagnosis rarely is made early and that the value of present surgical attack on this disease has fallen far short of being satisfactory. Surgical results have been disappointing, notwithstanding the fact that roentgenologic examination of the gallbladder has become one of the most accurate diagnostic laboratory procedures. Also, the anatomy of the gallbladder makes the organ easy to visualize and remove if removal is necessary. Why then should results of treatment of cancer of the gallbladder be so poor? There are two obvious reasons. The first is, that the insidious onset of symptoms is so typical of a benign cholecystic disease that not only the patient but also the physician is misled into believing that surgery is not especially urgent. This conclusion is to be expected, for most observers agree that the ratio of benign to malignant lesions of the gallbladder is approximately 100:1.[1, 2, 17] The second reason for the deadliness of this tumor is that it spreads in its early stages to the liver and the periportal structures.

During the past seven years, in private practice, we have encountered and treated 12 patients who had primary carcinoma of the gallbladder. We are reporting these cases not only to reiterate once more the need for early surgery in the patient with symptomatic gallbladder disease but also to recommend cholecystectomy for all patients with cholelithiasis unless there is a definite medical contraindication to surgery.

INCIDENCE

In Arminski's[2] thorough review of the literature, an analysis of 206,098 autopsies showed 908 cases of primary carcinoma of the gallbladder—an incidence of 0.43 per cent of all deaths and 4.53 per cent of all deaths from cancer. Tragerman,[17] in an analysis of 38,757 autopsies at the Los Angeles County Hospital, reported 135 cases of primary carcinoma of the gallbladder, an incidence of 0.35 per cent of all deaths and 2.11 per cent of all deaths from cancer. Arminski also reported that a cancer of the gallbladder will be found in 1.2 per cent of all operations for biliary disease. It is this small percentage that is responsible for the surgeon's low index of suspicion. In our small series, a cancer of the gallbladder was found in 2.8 per cent of 425 biliary operative procedures.

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SEX AND AGE

As would be expected, cancer of the gallbladder has a sex ratio of 3:1, with women predominating.[1] Also, by far the highest percentage of cases occur in patients from 40 to 80 years of age, the so-called cancer age group. Of our patients, the oldest was 76 and the youngest 47 years of age.

ETIOLOGY

As in most of the malignant lesions of man, no specific etiologic agent has been proved to initiate cancer of the gallbladder. The two conditions that are invariably present, however, are gallstones and inflammation.[18] More recently, cancer has been proved to arise from a papilloma.

Gallstones. Vast experimental work has been done to attempt to show that the gallstone is the specific agent that stimulates the transformation of a gallbladder into a cancerous growth. In 1947, Petrov and Kratkina[12] reported that cancer of the gallbladder developed in guinea pigs after glass beads had been introduced into the gallbladder. They successfully produced a cancer in 5 of these animals. Other work of a similar nature by Burrows[4] and also by Desforges[5] failed to show that foreign bodies in the gallbladder are definite carcinogenic agents. The low death rate from carcinoma of the gallbladder (0.45 per cent), when compared with the relatively high incidence of gallstones, also is evidence that stones are not the only etiologic factor to be considered. All authorities report a high incidence of gallstones in patients with this malignancy.[1, 2, 4] Arminski found that gallstones were present in 73.2 per cent of the patients he reviewed. Many authors report an even higher incidence. Gallstones were found in all of the 12 patients whose cases are reported in this study.

Inflammation. As gallstones and cholecystitis are closely associated, it is difficult to separate the two. Even in surgically removed gallbladders, with only microscopic evidence of carcinoma, inflammatory changes always were present. Ewing[6] believed that the combination of several factors, including gallstones, inflammation, and disturbed metabolism, may be the carcinogenic agents.

Papillomas. For many years, attempts have been made to incriminate the papillomas as a common precursor of cancer of the gallbladder; however, evidence supporting such a theory was scanty until the recent report of Tabah and McNeer,[16] who reported 4 cases of true papilloma of the gallbladder, 3 of which showed in situ carcinoma. True papilloma of the gallbladder, as differentiated from the cholesterol polyp, is a rare lesion. C. H. Mayo[11] in 1915 reported on 107 cases of papilloma in 2,538 surgically removed gallbladders. In 1929, Phillips[13] reported on 500 cases of surgically removed gallbladders, each containing a polyp. Closer study revealed that almost all of these papillomas really were cholesterol polyps. Such polyps are not true papillomas but rather a collection of cholesterol in a villus giving the appearance of a papillomatous-like growth.[16] These cholesterol deposits are detached easily from the gallbladder wall. The true papilloma is a distinct neoplastic growth consisting of a vascular connective tissue stalk covered by a single layer of columnar cells with repeated

rebranching to form a papillomatous growth.[16] Kerr and Lendrum[9], in 1936, reviewed the literature and found only 11 cases in which the *growths* were true papillomas. Shepard,[15] in reviewing surgically removed gallbladders at the Mayo Clinic, collected 45 cases of papilloma and estimated that roughly 1 in every 100 surgically removed gallbladders contained a papilloma.

These papillomas cause symptoms similar to those in the typical case of cholecystitis: pain in the upper right quadrant, dyspepsia, and intolerance to certain foods. They can be diagnosed with a high degree of accuracy by cholecystography, as the defect produced by the papilloma maintains a constant position, while stones will gravitate to the most dependent portion of the gallbladder.

The old belief that papillomas had no precancerous tendencies now is generally recognized as misleading, for the large majority of these lesions were cholesterol polyps and not true papillomas. The patient with a papilloma should be treated by cholecystectomy, not only because of the accompanying symptoms, but also because, as Tabah and McNeer[16] believe, the papilloma is a definite precancerous lesion with real danger of undergoing malignant degeneration.

SYMPTOMATOLOGY

The symptomatology of cancer of the gallbladder is that of cholecystitis and cholelithiasis. Pain in the right upper quadrant, dyspepsia, and intolerance to some foods are the usual presenting symptoms. Even in cases later shown to be cancer, rarely are any unusual symptoms elicited. This lack of a distinguishing symptom has helped to prevent an early clinical diagnosis. When loss of weight, anemia, progressive jaundice, and a palpable mass are present, one invariably finds an extensive, incurable cancer.

More than 70 per cent of patients found to have a cancer of the gallbladder have a history of longstanding biliary disease. Three of our patients had symptoms of less than one year. Seven of them had had infrequent episodes of biliary distress for more than four years.

Pain. Pain in the upper right quadrant of the abdomen is the most common complaint. It is present in more than 70 per cent of the patients. It is dull and intermittent and often is related to the intake of food. In the later stages, the pain becomes a constant *boring* type. It was the presenting symptom in 9 of our 12 patients.

Dyspepsia. Eructation, bloating, and difficulty with digestion is found in about 70 per cent of these patients. These are the common symptoms of cholecystitis and would be expected to be present, as evidence of inflammation always accompanies a malignancy of the gallbladder. Eleven of our 12 patients complained of such symptoms.

Weight Loss. The loss of weight is a sign that usually is associated with any malignancy of the digestive system. The location of the lesion, its rate of growth, and the length of its duration are the governing factors in this loss of weight. Many patients with benign gallbladder disease may lose weight, but the rapid loss of weight by a patient who has had known gallbladder disease for several years always suggests a malignancy. Ten of our 12 patients complained of rapid loss of weight.

Jaundice. Of 1,062 patients who had cancer of the gallbladder, 57.7 per cent were jaundiced.[2] In these patients, the jaundice was progressive. It was shown to be caused by compression of the common or hepatic ducts by an enlarged quadrate lobe of the liver or by metastatic lymph nodes in the periportal tissues. Jaundice was a finding in only half of our patients.

Palpable Mass. Most reports show that a palpable mass is present in more than half of these patients. This finding is only further proof that the lesion is diagnosed late, for we have found no report of a patient with the palpable mass of a gallbladder carcinoma who has survived two years. This mass usually is tumor tissue and not just a dilated gallbladder. A palpable mass was present in 4 of our patients.

Laboratory Findings. The laboratory is of little aid in diagnosing this disease, but it helps evaluate the general condition of the patient. An anemia or a leukocytosis may be found. The jaundice is of the obstructive type. The cholecystogram may show the presence of a stone, but more commonly it shows a nonfunctioning gallbladder. The ingestion of barium may show a biliary fistula or a pressure defect in the duodenum. These findings are further evidence of a malignant process.

TREATMENT

Early surgical removal of the cancerous gallbladder is the only satisfactory treatment. The reported cases of a five year cure, with few exceptions, are patients whose gallbladders were removed for cholelithiasis; the pathologist later found a small site of malignancy. One notable exception is a case reported by Booker and Pack.[3] The patient was alive five years after the removal of a gallbladder showing adenocarcinoma, grade III, with cystic duct lymph node metastases. If the patient is jaundiced or if a palpable mass is present, the surgeon often can do no more than a cholecystostomy. Radical procedures for this malignancy have not been encouraging. Sheinfeld[14] reported the cases of 36 patients who were treated by cholecystectomy and partial hepatectomy. Good palliative results were obtained in 19.4 per cent of the patients. The long-term results are not available, as many of these cases were collected from the literature and had not been followed closely. Even this figure, however, compares favorably with the palliation following cholecystectomy, which was only 6.7 per cent in 417 cases also included in this report.

Nine of our 12 patients were treated by cholecystectomy, 2 by cholecystectomy with partial hepatectomy, and 1 by cholecystostomy alone. Five of these patients were dead within three months, another 5 died between three and 12 months following operation, and 2 patients are still alive after one year. One of these last 2 patients has recently developed symptoms of a recurrence following a cholecystectomy and partial hepatectomy which were done 16 months ago. The other patient is alive and apparently well two years after a cholecystectomy done for cholelithiasis. At operation there was no gross evidence of tumor; yet, microscopic sections showed a well localized, infiltrating adenocarcinoma.

PATHOLOGY

There are two types of cancer of the gallbladder, adenocarcinoma and epidermoid carcinoma. More than 95 per cent are of the adenocarcinoma type. There is a wide difference of opinion regarding the epidermoid variety. Some authors believe that they represent a form of squamous metaplasia; others believe they are not true squamous cells but merely altered adenocarcinoma cells.

There are three types of adenocarcinoma recognized grossly:

1. Infiltrating carcinoma: This scirrhous carcinoma infiltrates the gallbladder wall and causes it to be greatly thickened and firm. The gallbladder may be contracted and the lumen may be almost obliterated. There is an abundance of connective tissue elements intermingled with the rapidly growing carcinoma cells. This type is present in about 70 per cent of the patients.

2. Papillary carcinoma: This tumor appears to arise from a localized mucosal area and it has a tendency to grow within the lumen, where it forms a large, bulky mass. It is found in about 20 per cent of the patients.

3. Colloid carcinoma: This type represents an overgrowth of all mucosal elements and results in a soft, necrotic mass. This friable tumor causes a perforation of the gallbladder wall more often than the other types.

The gross pathology of cancer of the gallbladder is quite difficult to recognize unless the tumor is far advanced. The thick-walled gallbladder differs very little from that of chronic cholecystitis. Recently, Marks[10] has observed that in cancer of the gallbladder, the hepatoduodenal ligament usually is a brawny, densely thickened structure. In his experience, this thickening was far greater than could be expected from the inflammation of cholecystitis. In 2 of our most recent patients, we observed this brawniness of the hepatoduodenal ligament, and we will continue to examine this structure closely to evaluate Marks' interesting report further.

Cancer of the gallbladder may spread by local extension, by lymphatic channels, and by the hematogenous route. It involves the liver bed early, both by local extension and also by the subserous lymphatic plexus. The lymphatic channels and nodes along the cystic duct and periportal structures are involved early. True hematogenous spread occurs but is rather uncommon.

COMMENTS

The uniformity of poor results reported in the literature and our own poor results influence us to strongly recommend cholecystectomy to patients shown to have gallstones if there is no medical contraindication for surgery. We recommend it even though their symptoms may be mild or even absent.

In advocating cholecystectomy for these patients, one must remember that the greatest factor to consider is the possibility of death from the benign complications of cholelithiasis. Even though the operative mortality rate for cholecystectomy is slightly higher than the incidence of cancer of the gallbladder, the physician must consider the deaths from pancreatitis, acute cholecystitis,

common duct obstruction, and cholangitis. Jaguttis[7] reported 114 cases of cholelithiasis over a period of 10 years. Of the patients who died, 41 per cent were dead from some complication of cholelithiasis. Other authors[8] have reported on autopsy series which showed that complications of gallstones caused the deaths of 12 per cent of the persons having such stones. The decision for cholecystectomy in the asymptomatic patient should be based, therefore, on the protection against the development of these complications rather than merely for prophylaxis against the development of cancer.

SUMMARY

Despite new and radical surgical technics, cancer of the gallbladder continues to be a fatal disease in all but a few patients. Its early involvement of vital structures precludes the possibility of a cure if the lesion has progressed sufficiently to be recognized grossly as a malignancy. Its symptoms so closely resemble those of the much more common benign inflammatory condition of the gallbladder that the clinician has no chance of diagnosing a cancer of the gallbladder except in the late stages of the disease.

With the presence of gallstones or a papilloma as evidence of a diseased gallbladder, we believe that the patient should have a cholecystectomy, despite a lack of symptoms, unless a definite medical contraindication exists. When the high risk from the complications of gallstones is combined with the low incidence of the development of a malignancy, it overweighs the small operative risk of cholecystectomy.

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FASCIAL SPACES OF THE NECK

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The description and the nomenclature which follow are based primarily upon the teaching of Doctor Henry James Prentiss who was Professor of Anatomy at the University of Iowa when I began the study of medicine. The limitation of time would prevent my discussing specific surgical conditions which may be influenced by the disposition of the cervical fascias, even if I were qualified to do so. I shall, therefore, confine myself to purely anatomic considerations. You, who are surgeons, are in a much better position than I to talk of the practical applications of anatomy.

Dr. Melvin A. Casberg[1] has emphasized the importance of the fascial planes of the neck and has stated that the clinician who understands the structure and disposition of these fascias is in possession of basic diagnostic and therapeutic information which "raises him above the level of empiricism." Dr. Casberg has gone on to list the bases for clinical significance of the cervical fascial planes as follows:

1. Their function as directives or limitants in the spread of extravasations, whether these be inflammatory or hemorrhagic;
2. Their aid as diagnostic and therapeutic landmarks;
3. Their role in the support of adjacent structures;
4. And their influence on the direction of expansion of neoplasms, whether these be benign cysts or malignant tumors.

An appreciation of the general disposition of the cervical fascias is best gained from a cross section at the level of the fourth, fifth, or sixth cervical vertebra. In a cross section at the level of the body of the fourth vertebra (fig. 1), the deep or enveloping fascia is seen to lie beneath the superficial fascia and the platysma muscle; the muscle is placed deeply in the superficial fascia. As a matter of fact, the superficial fascia can be divided into a superficial or fatty layer and a deep or membranous layer and it is in the latter that the platysma muscle is found.

Usually, the deep or enveloping fascia is described as though it encircled the neck from the anterior midline to the posterior midline. A more reasonable description seems to me to consider the fascia as encircling the neck in a forward direction from an attachment to the ligamentum nuchae, with fusion of the fascias of the two sides in the anterior midline. As the fascia leaves the ligamentum nuchae on either side, it divides into two layers to enclose the trapezius muscle. At the anterior border of the trapezius, the two layers fuse and cover the posterior triangle of the neck and then again divide to envelop the sterno-

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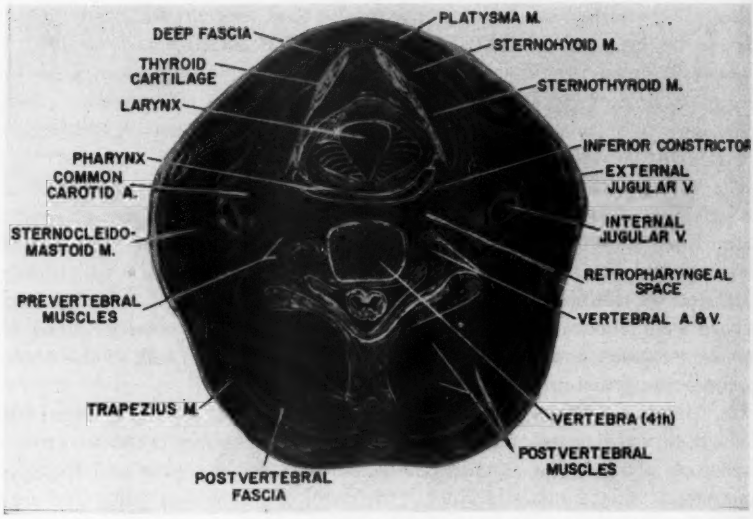

FIG. 1. Cross section of the neck at the level of the body of the fourth vertebra. The figure is a black and white reproduction of a colored lantern slide which was prepared by photographing the author's blackboard reproduction of section 18 in Eycleshymer and Schoemaker's "Cross-Section Anatomy."

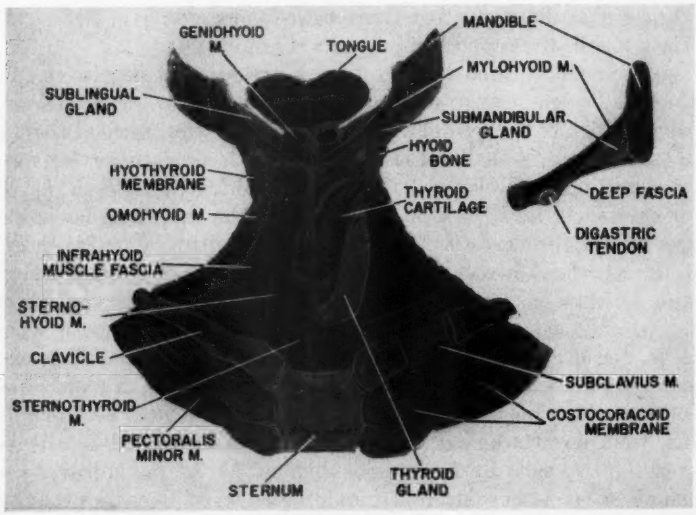

FIG. 2. Diagrammatic representation of the infrahyoid muscle fascia and the disposition of the deep fascia in the suprahyoid region. This figure is a reproduction of a colored lantern slide prepared from a blackboard drawing by the author. On the left side of the neck a portion of the infrahyoid muscle fascia has been removed to expose the location of the larynx and thyroid gland. On the left side a segment of the clavicle has been removed in order to demonstrate the continuity of the infrahyoid muscle fascia and the costocoracoid membrane.

cleidomastoid muscle. In the area anterior to the anterior border of the sternocleidomastoid, on either side, the deep fascia covers the anterior triangle.

Superiorly the deep fascia is attached to body and ramus of the mandible (fig. 2), to the periosteum overlying the mastoid process, and to the superior nuchal line. Inferiorly its attachments are to the sternum and clavicles and to the acromion processes and spines of the scapulas. Approximately midway between its upper limit and its inferior limit the deep fascia is fused with the periosteum of the hyoid bone and with the fascia immediately overlying the larynx (fig. 3).

Certain reflections of the deep fascia, in the suprahyoid region, are of clinical significance. Anteriorly, these reflections result in the separation of the submental and submandibular spaces (fig. 2). This separation is brought about by the fact that the deep fascia, as it passes upward from the hyoid bone to the body of the mandible, divides, on either side, to envelop the anterior belly of the digastric muscle. The interval between the digastric muscles, which is covered superficially by the deep fascia proper and whose deep boundary is that reflection of the deep fascia which forms the muscle fascia of the mylohyoid muscle, constitutes the submental space and contains the submental lymph nodes. As the deep fascia of the submandibular region extends from the upper border of

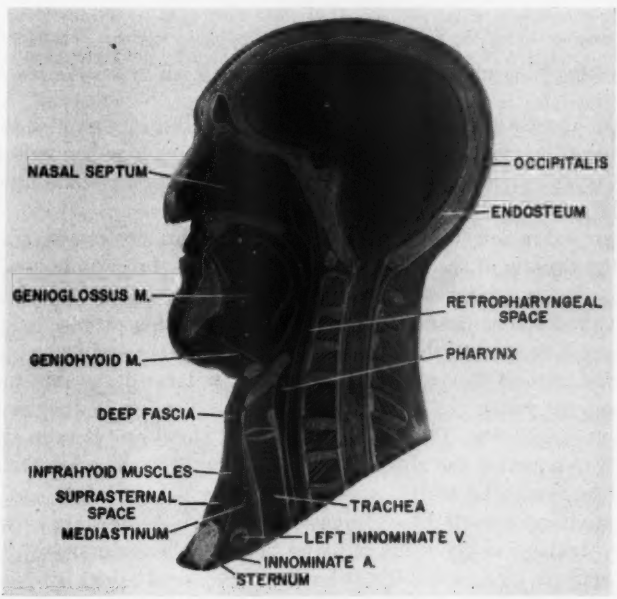

FIG. 3. Median sagittal section of the head and neck to show the relationships and extent of the retropharyngeal space, the suprasternal space (of Burns) and the cervical extension of the middle mediastinum. This figure is a black and white reproduction of a colored lantern slide made from a blackboard drawing by the author modified from a figure appearing on page 63, "Atlas of Human Anatomy" by Barry J. Anson.

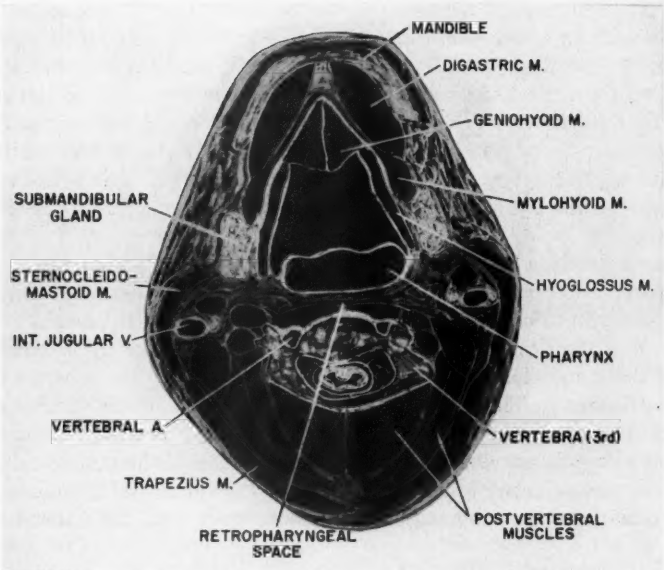

FIG. 4. Cross section of the neck at the level of the third vertebra. This figure is a black and white reproduction of a colored lantern slide prepared by photographing the author's reproduction of section 16 in Eycleshymer and Schoemaker's "Cross-Section Anatomy."

the digastric muscle (and its tendon) to the mandible, it separates into superficial and deep layers. The deep layer covers the mylohyoid muscle while the superficial layer covers the submandibular fossa; the intermediate layer divides to form the capsule of the submandibular gland.

In the parotid region (fig. 4), the deep fascia divides into two layers and thus provides the capsule of the gland. The deep layer of the capsule is a reflection from the posterior border of the ramus of the mandible to the styloid process and to the stylohyoid ligament and, in turn, from this process and ligament to the mastoid process and the anterior border of the sternocleidomastoid muscle. The anterior part of this reflection sometimes is termed the stylomandibular ligament or membrane (fig. 5) and the posterior portion is designated as the stylomastoid membrane. The superficial layer of the deep fascia in this region, in addition to covering the gland, extends upward over the masseter muscle to attach to the zygomatic arch.

The parapharyngeal or pharyngomaxillary space, in the area immediately inferior to the base of the skull, is found between the stylomastoid membrane and the pharynx (figs. 4 and 5). It frequently is involved in extensions of inflammatory processes from the tonsils and pharynx. The superior pharyngeal constrictor is said to form the medial boundary of this potential space. The space extends forward as well as downward so that, ultimately, the submandibular gland and the fascial layers making up the carotid sheath form the posterior

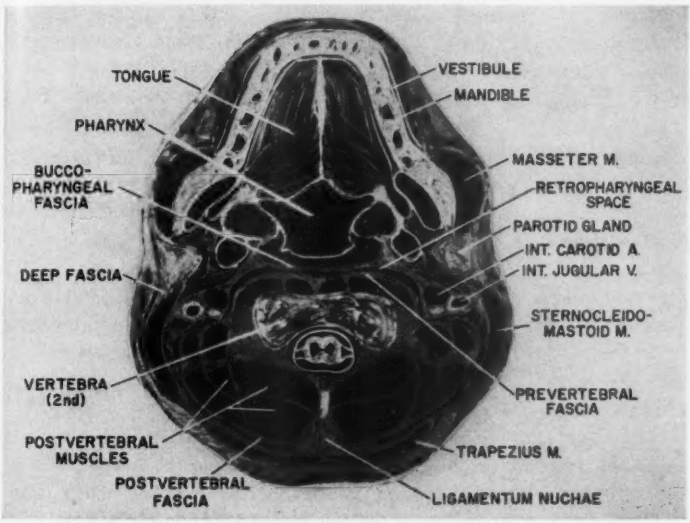

FIG. 5. Cross section through the neck at the level of the body of the second vertebra. This figure is a black and white reproduction of a colored lantern slide made from the author's blackboard reproduction of section 14 in Eycleshymer and Schoemaker's "Cross-Section Anatomy."

boundary and the internal pterygoid muscle contributes to its lateral boundary. Superiorly, the space is limited by the base of the skull and inferiorly it is said to be adjacent to, but not necessarily continuous with, the carotid sheath.

There are three major reflections of the deep or enveloping fascia of the neck (fig. 6). The first of these extends between the superior and inferior limits of the neck but is actually continuous with the vertebral fascia throughout the length of the vertebral column. An intermediate reflection extends throughout the cervical region and continues into the mediastinum; an anterior reflection is confined to the infrahyoid region of the neck and is continuous with an important fascial layer in the pectoral region.

The vertebral reflection of the deep fascia of the neck is well shown in a section through the neck at the level of the fifth vertebra (fig. 6). It separates from the enveloping fascia immediately lateral to the ligamentum nuchae and covers the postvertebral muscles. Anteriorly it is attached to the posterior tubercles of the transverse processes of the vertebrae. From this point it is reflected forward to the anterior tubercles and then medially in front of the prevertebral muscles. The cervical nerves emerge from the spinal canal through the intervertebral foramina and into the interval between the anterior and posterior tubercles of the transverse processes where they are covered by the vertebral fascia. In the lower part of the neck those cervical nerves which contribute to the brachial plexus and which unite to form the upper, middle and lower trunks of that plexus lie beneath the vertebral fascia as it extends from posterior to anterior tubercles. That portion of the vertebral fascia extending between the anterior

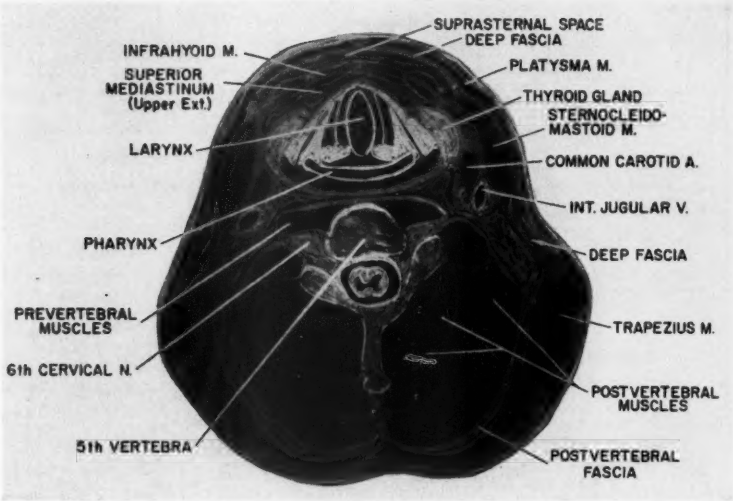

FIG. 6. Cross section through the neck at the level of the body of the fifth cervical vertebra. This figure is a black and white reproduction of a colored lantern slide prepared by photographing the author's modified reproduction of section 19 of Eycleshymer and Schoemaker's "Cross-Section Anatomy."

tubercles of one side and those of the opposite side is almost universally referred to as *prevertebral*. That portion extending from the anterior tubercles to the ligamentum nuchae on either side is less commonly but appropriately referred to as *postvertebral*. As mentioned above these fascias are continuous with the prevertebral and postvertebral fascias throughout the length of the vertebral column.

At the base of the neck, the vertebral fascia is carried to the first rib with the insertions of the anterior and middle scalene muscles. A process of the fascia is carried outward in the interval between these two muscles by the nerves of the brachial plexus and the subclavian artery which at this point becomes continuous with the axillary artery. Some surgical anatomy texts attribute considerable significance to this process of vertebral fascia and state that abscesses resulting from infectious processes in the vertebral bodies may extend downward beneath the prevertebral fascia and then follow the axillary process of this fascia into the axilla. Casberg attributes little significance to the continuity between the prevertebral fascia and the axillary sheath.

The intermediate reflection of the deep or enveloping fascia is a two-layered structure which forms the carotid sheath and then continues medially to form the visceral fascia. The visceral fascia encloses the pharynx, esophagus, larynx, trachea and thyroid gland. Its anterior layer often is referred to as the pretracheal fascia and its posterior portion as the buccopharyngeal fascia. Casberg has designated both the anterior and the posterior portions of the visceral fascia as *pretracheal leaflets*. I cannot visualize a fascia which covers the posterior

surfaces of the pharynx and esophagus as *pretracheal* and I must maintain that such a designation adds to rather than detracts from the confusion which exists with respect to the many terminologies which have been applied to the cervical fascias. The visceral fascia continues into the middle mediastinum where it covers the great vessels, pericardium, and esophagus.

The carotid sheath, in the lower part of the neck, contains the internal jugular vein, the vagus nerve and the common carotid artery. Above the hyoid level it is continuous with the internal carotid division of the common carotid artery and, at this level, also contains the internal jugular vein and vagus nerve. At the upper limit of the carotid sheath the vagus nerve has associated with it the glossopharyngeal, the spinal accessory and the hypoglossal nerves, each of which leaves the sheath at a relatively high level. The double-layered fascial connection between the carotid sheath and the visceral compartment of the neck is important because it serves as a bridge through which the nerve and blood supply to, and the return circulation from, the visceral structures are effected.

A clinically significant potential space is developed between the prevertebral and buccopharyngeal fascias; this is designated as the retropharyngeal space and it may be the site of retropharyngeal abscesses, secondary to nasal or nasopharyngeal infections. The lymph nodes which drain the nasopharyngeal region are found in the upper part of the retropharyngeal space. The symptoms (dyspnea, dysphagia and dysphasia) which result from this type of abscess may be simulated by accumulations of pus beneath the prevertebral fascia which account for forward bulging of it.

The anterior reflection of the deep or enveloping fascia (fig. 5) can be appropriately designated as the infrahyoid muscle fascia. It can be so-called because it envelops and forms the fascia of the sternothyroid, sternohyoid and omohyoid muscles. An interval developed between it and the deep fascia proper is called the suprasternal space (of Burns). The upper limit of this space is at the point where the deep fascia and infrahyoid muscle fascia separate in front of the larynx (fig. 3) and its lower limit is the superior border of the sternum and, to some extent, the superior surfaces of the clavicles. The enveloping fascia fuses inferiorly with the periosteum overlying the anterior surfaces of the sternum and clavicles while the infrahyoid muscle fascia fuses with the periosteum covering their posterior surfaces. The anterior jugular veins and some lymph nodes are found in this space. It is of considerable significance that the infrahyoid muscle fascia divides around the subclavius muscle and then is continuous into the pectoral region as the costocoracoid membrane which envelops the pectoralis minor muscle and thus contributes to the anterior wall of the axilla.

The interval which is developed between the pretracheal fascia and the infrahyoid muscle fascia is particularly important because it represents a direct upward extension into the neck of the superior mediastinum (fig. 3). Because the heart develops in the cervical region and descends into the thorax, carrying with it the great vessels, there always is the possibility that these vessels may lie at a higher than usual level such as to place them in the cervical extension of

the superior mediastinum. In such cases they may present a considerable hazard to surgery in the cervical region.

CONCLUSIONS

In conclusion it may be reiterated that localization or extension of neck infections may depend upon the fascial spaces formed by reflections of the deep or enveloping fascia. The chief reflections are three in number: vertebral, enclosing the vertebral column and the muscles in immediate relation to it; visceral, forming the carotid sheaths and surrounding the larynx, pharynx, trachea, esophagus and thyroid gland; and infrahyoid, enveloping the infrahyoid or strap muscles. In the suprahyoid region the reflections of the deep fascia account for separation of the area into submental, submandibular, and parotid regions. Retropharyngeal and parapharyngeal abscesses are particularly likely to occur in the cervical region and they tend to remain localized because of the disposition of the fascias.

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GASTRIC POLYPOSIS WITH CASE REPORTS

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RICHARD E. BOWER, M.D.

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Gastric polyposis is a relatively rare disease. In 1926 Pearl and Brunn[7] collected 79 cases from the literature between 1820 and 1926 and added 5 of their own. From 1926 to 1941 they found 38 additional cases and added 3 of their own, making a total of 125 cases reported. Balfour[1] states that in 69,000 celiotomies done at the Mayo Clinic, of which 8,000 were primarily gastric operations, only 1 case of gastric polyposis was encountered. The general incidence ranges from .25 per cent found by Spriggs and Marxer[8] in 4,400 autopsies to .7 per cent in a review of 7,000 autopsies by Lawrence[4]. Yarnis and associates[9] found only 8 cases in 8,735 routine autopsies. Thirty of the last 41 cases reported by Pearl and Brunn occurred in men. The youngest patient was 23 and the oldest was 80 years of age. Two-thirds of the cases were discovered in the fourth and fifth decades. We are reporting 2 cases recently encountered in our practice.

Gastric polyposis still is a confusing entity although much has been written about it recently. The term polyp has been used to describe any pedunculated or sessile tumor arising from the mucosa and projecting into the lumen of a viscus. This describes the physical characteristics of the growth without reference to its histologic character. We use the term gastric polyposis in this paper to describe three or more polyps of the stomach. In 1888 Menetrier[6] divided these tumors into two types. The first type *polyadenomes polypeaux* consisted of polyps with independent attachments, either pedunculated or sessile. Microscopically the submucosa and muscularis extended out into the stalk. Cyst formation was common. (Both cases which we are reporting conform to this type.) The second type which he described as *polyadenoma en nappe* was a slightly elevated, well demarcated plaque, not cystic or pedunculated, composed of closely placed folds of hypertrophic mucous membrane. Menetrier was of the opinion that both types were the result of chronic inflammation. Boyd[2] stated that gastric polyps may be either neoplastic or inflammatory in nature. In most cases the nodules are adenomatous, that is, neoplastic. They may be scattered over the entire mucosal surface but often are localized to one area, usually the greater curvature and the lower third of the stomach. Microscopically there is an orderly arrangement of the new formed glands, many of which show cystic dilatation. Boyd also pointed out that in addition to multiple adenomata there may be a hypertrophic condition of the gastric mucosa which is thrown into great folds resembling the convolutions of the brain. He regarded this as a benign inflammatory lesion and probably related to hypertrophic gastritis. In 1 patient examined by him the thickened mucosa was due largely to edema.

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There is much controversy over the etiology. Pearl and Brunn stated that there are two recognized theories regarding the origin of polyposis of the stomach. One group, including Wechselmann and Rippert, is of the opinion that they are of congenital origin—true neoplastic growths. However, in their series of 125 cases Pearl and Brunn did not find a single instance in which a familial predisposition was evident. The second group, in which are Cornil, Menetrier, and Verse, stated that the growths are due to chronic inflammation. They quoted Konjetzny as claiming that he found all stages in the chain from hypertrophic gastritis to adenoma, and finally to carcinoma.

There is a great difference of opinion in the literature concerning the incidence of malignant degeneration occurring in gastric polyposis. It appears that gastric polyps do not degenerate into cancer as frequently as do polyps of the rectum and colon. However, Pearl and Brunn reported malignant degeneration in 19 of 37 cases. Lawrence[4] found malignant degeneration in 3 of 50 cases. Marshall[5] reported 2 of 3 cases with malignant degeneration. It is evident that until a larger series of cases accumulate in the literature the incidence of malignant degeneration will remain in controversy.

SIGNS AND SYMPTOMS

There are no symptoms which are characteristic of this disorder. Epigastric pain or distress, nausea and vomiting seem to be the most frequent symptoms. Hematemesis occurs frequently. Sometimes it is an exsanguinating hemorrhage, even to the point of causing death. Bleeding, either gross or occult, is present in between 50 to 60 per cent of the patients. Large pedunculated tumors seem to cause more trouble than the sessile type, especially when they are located near the pylorus. They may prolapse into the duodenum causing profuse vomiting and at times gastroduodenal intussusception. They also seem to produce more bleeding than the sessile type of tumor due to ulceration and avulsion of the tumors from the stomach wall. Tenderness is present occasionally over the epigastrium and rarely a mass may be felt.

DIAGNOSIS

This disease may remain symptomless for years and it may never be diagnosed during life. One cannot always rely upon the roentgenogram to make the diagnosis of gastric polyposis. Hypertrophic gastritis, carcinoma, sarcoma, food particles, bezoars, and blood clots in the stomach may be difficult to differentiate roentgenologically. Brunn and Pearl, in their series of 41 personal and collected cases, stated that the correct diagnosis was made by roentgenogram in 17 patients, by operation in 13, by examination of the excised surgical specimen in 5, and by autopsy in 6. In both cases which we are reporting two gastrointestinal roentgenograms were made to be sure the deformity of the stomach was constant before making the diagnosis. The constant deformity enabled us to make the preoperative diagnosis of polyposis in both cases on the roentgenographic findings. As the gastroscope becomes used more universally the diagnosis of gastric polyposis probably will be made more frequently and more accurately.

In the laboratory findings it has been noted that achloryhdria more often is present with gastric polyposis than with carcinoma of the stomach. There usually is occult or frank blood in the gastric washings and the stools, with a secondary anemia. At operation the surgeon may have difficulty in diagnosing gastric polyposis in the unopened stomach. He may feel nothing abnormal; the gastric wall may be thickened; the stomach may feel as though it contained worms; or there may be several discrete tumors. A gastrotomy is necessary for accurate diagnosis.

TREATMENT

The treatment of gastric polyposis is surgical exploration and excision. A gastrotomy is necessary to determine the character and extent of the lesion. By inspecting the gastric mucosa the line of excision is determined. If the area of the stomach involved is small and the tumors are found to be benign by immediate pathologic examination, local excision, including the entire thickness of the gastric wall, may suffice. However, if a large area of the stomach is involved or if there is any question of malignancy a high subtotal gastrectomy is indicated. Doyle[3] recently reported 1 case in which a total gastrectomy was necessary to remove the entire tumor bearing area. He found 9 other similar cases reported in the literature in which total gastrectomies were done.

CASE REPORTS

Case 1. Mrs. C. R. W., aged 39, was admitted to the hospital on Dec. 23, 1950 and discharged on Jan. 21, 1951. She was six months pregnant. Her chief complaints were severe headaches and swelling of her feet and ankles for one week. There had been no vomiting during her pregnancy or at any other time. For three to four years she had occasional epigastric cramping which lasted only a few minutes and was not severe. There had been no hematemesis or tarry stools.

She was a well nourished white woman. The blood pressure was 150/90, the pulse was 84 per minute, and the temperature was 98.6 F. There was a small recurrent nodular goitre. The abdomen was normal except for the uterus which was six months pregnant. There was a moderate pitting edema of the lower extremities. The red blood count was 2,480,000 per cu. mm. and the hemoglobin was 50 per cent. The white blood count was 4,600 per cu. mm. with 58 per cent polymorphonuclear cells. The blood serology was negative. The urine had a specific gravity of 1.029, 500 mg. albumin, 3 to 4 granular casts, 1 to 2 hyaline casts, 2 to 3 red blood cells, and 6 to 8 pus cells per high power field. The urine was negative for sugar. The non-protein nitrogen was reported 44 mg. per 100 cc.

The admitting diagnosis was pregnancy, six months, complicated by pre-eclampsia with anemia. She was given the usual treatment for these conditions and responded well. Three days after admission she vomited approximately 1000 cc. of blood which produced profound shock, this was treated by multiple blood transfusions. She was placed on an ulcer regimen and no further gross bleeding occurred. A few days later a gastrointestinal roentgenologic study was as follows: "The rugae in the stomach are very suggestive of a diffuse malignancy or polyposis. These findings may represent blood clots in the stomach. Repeat study in a few days." The second study revealed the same findings (fig. 1). On this basis a preoperative diagnosis of gastric polyposis was made. A laparotomy confirmed the roentgenographic findings. Complete exploration of the abdomen showed everything normal except a six months pregnancy and gastric polyposis. Numerous pedunculated and sessile tumors could be palpated throughout the distal two-thirds of the stomach. The larger pedunculated tumors were located in the pyloric region. There was no gross evidence of cancer seen. A

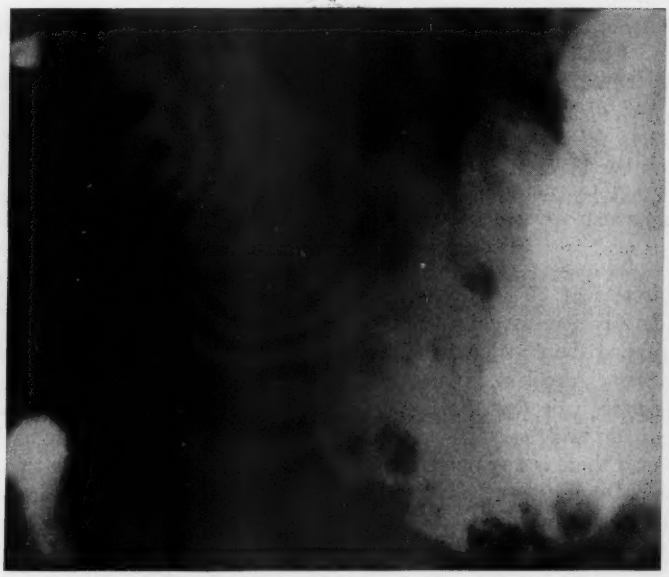

FIG. 1. Polypoid defects in the barium filled stomach

gastrotomy was done in order to permit inspection of the mucosa. A subtotal gastrectomy was done. Eighty per cent of the stomach was removed. Continuity was restored by a gastrojejunostomy placed anterior to the colon.

The pathologist's report was as follows: "The specimen consists of a segment of stomach measuring 15 cm. in length on the lesser curvature. The mucous membrane is covered with polypoid tumors which measure up to 2 cm. in greatest diameter. Many of these tumors are pedunculated but others are just bulges on the mucous membrane (fig. 2). Microscopic: The muscularis of the stomach appears thin and edematous. Brunner's glands are numerous and appear normal. The mucous membrane is thrown up in papillomatous and polypoid masses. The connective tissue cores in these polypi are composed of loose connective tissue which is very vascular. The epithelial cells in these areas generally are swollen and ballooned up with secretory material. There is a diffuse leukocytic infiltration all through the stomach with plasma cells predominating. Diagnosis: Multiple gastric polypi."

Her recovery was uneventful except for a spontaneous abortion of a macerated fetus on the eighth postoperative day.

She has been followed closely over the past three years and has remained symptom free, with no evidence of anemia. Several postoperative gastrointestinal roentgenologic studies have not shown any evidence of recurrence.

Case 2. W. R. W., a colored man, aged 58, was admitted to the hospital on Sept. 21, 1953 and discharged on Oct. 7, 1953. He was referred to the hospital for repair of a left inguinal hernia. There was no voluntary history of gastrointestinal symptoms. By asking leading questions we were able to obtain a history of occasional indigestion and possibly an occasional tarry stool.

He was a well nourished colored man. The blood pressure was 190/100, the pulse was 68 per minute, and the temperature was 98.6 F. The mucous membranes were pale. The abdomen was normal except for a small left indirect inguinal hernia. Laboratory findings were as follows: Red blood count was 2,910,000 per cu. mm. with 64 per cent hemoglobin, hematocrit 46 and color index 1.1. White blood count was 3,900 per cu. mm. with 60 per cent polymorpho-

FIG. 2. Excised segment of stomach showing multiple polyps. The larger polyps are in the pylorus.

nuclear cells. A blood serology study was negative. Urinalysis and stool examinations were normal. A rectal and sigmoidoscopic examination was normal. A barium enema revealed an occasional diverticulum.

A gastrointestinal roentgenogram was reported as follows: "The gastric rugae are rather heavy throughout and there appear to be multiple polypi in the region of the pars media. Suggest re-examination in a few days." The second roentgenogram showed the same findings (fig. 3). A preoperative diagnosis of gastric polyposis was made. Surgical exploration of the abdomen showed a small left inguinal hernia and numerous gastric polypi starting about 6 cm. above the pylorus and extending proximally 10 cm. A gastrotomy was done. Direct inspection of the mucosa showed eight sessile polyps scattered over large swollen rugae. There was no gross evidence of malignancy. A sleeve resection was done. Sixty per cent of the stomach was removed.

The pathologist's report was as follows: "The specimen is a segment of the stomach 20 cm. in length. The wall of the stomach is uniform in thickness but there are six or eight raised dome shaped tumors bulging into the lumen. The largest of these measures 2 cm. in its greatest dimension. I do not see any extension of the tumor into the muscle and I do not find any enlarged lymph nodes in the attached mesentery (fig. 4). Microscopic: The muscle and connective tissue layers of the wall of the stomach are normal. The subepithelial connective tissue peaks out under the tumors and the mucosa remains limited by the basement membranes. The mucosa is hypertrophied and it is in long finger-like processes. I do not see malignant change. Diagnosis: Multiple gastric polypi."

Following the operation he made an uneventful recovery. He has maintained his weight. The anemia has not recurred. A gastrointestinal roentgenogram made approximately two months after operation did not show evidence of recurrence of the polypi.

FIG. 3. Polypoid defects in the barium filled stomach (fluoroscopic spot film)

FIG. 4. Excised segment of stomach showing polyps

CONCLUSION

Gastric polyposis relatively is a rare disease. It must be considered in gastric hemorrhage and in unexplained hypochromic anemia. Malignant degeneration occurs frequently. The treatment is surgical excision. Two cases of gastric polyposis in which the patients were treated by partial gastrectomy are reported.

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POSTOPERATIVE HYPOTENSION ASSOCIATED WITH HYPOKALEMIA DUE TO CORTISONE THERAPY: A CASE REPORT

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Hypotension during an operation or in the immediate postoperative period is common. It frequently is unexpected and therefore demands immediate explanation. Intelligent therapy depends upon the accuracy of the explanation. In general, the poorer the risk, and the more extensive the surgery, the more often hypotension will occur as a complication of the operation. Fortunately, the majority of episodes of such hypotension have a simple and obvious explanation. The most common cause is an ineffective blood volume. This, in turn, usually is due to the combination of uncompensated blood loss with generalized vasodilatation due to the anesthetic. Other causes, however, such as excessive anesthesia, myocardial infarction, cardiac decompensation, adrenal insufficiency, unrecognized hypothyroidism, hyponatremia, neurogenic vasodilatation, transfusion of mismatched blood, or improper medication may be more difficult to identify.

The poor response of the patient with Addison's disease to trauma, including surgery, narcotics, general anesthesia, and infection is well known. Hypotension is a prominent clinical feature of this poor response. Rarely are patients with subclinical adrenal insufficiency encountered, who also tolerate an operation poorly. Recently Thorn and associates[15] and Salassa and associates[14] have pointed out that after prolonged treatment, even with moderate doses of cortisone, a profound depression of adrenocortical function may develop. Operative trauma inflicted during this depression may prove disastrous.

That prolonged cortisone therapy may lead to potassium depletion also is well known. Little attention, however, has been called to the possible relation of potassium depletion to hypotension.

Friedman and associates[7] and Freed and Friedman[6] have shown in rats that potassium deficiency is correlated with hypotension. They also found that the administration of potassium would restore the lowered pressure to normal. They concluded that the hypotension noted in the rat after potassium deprivation appeared to be due to changes produced in the peripheral vasculature, functional in nature, and quickly reversible after the administration of potassium alone. Clinically, Lans, Stein and Meyer[8] showed in an analysis of 404 patients with hypokalemia—of which 241 were postoperative—that anorexia, nausea, muscle weakness, loss of tendon reflexes, mental depression, lethargy, apathy and drowsiness usually were present; mental confusion, shallow respiration, abdominal distension, paralytic ileus, irregular pulse and a fall in blood pressure

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frequently were present; and complete muscle paralysis, delirium and hallucinations occasionally were present. They did not mention the frequency of coexisting hyponatremia.

Evans,[5] Marks,[9] Patton and associates[12] and Lans, Stein and Meyer have found in their clinical studies that the commonest cause of hypokalemia in surgical patients is the loss of gastrointestinal content combined with a low or absent intake of potassium. Randall[13] and others have emphasized the importance of the postoperative loss of potassium. Berry[1] determined the urinary output of potassium for each of five 6 hour periods for the first 30 hours following major operations in 10 patients and found that the excretion was greatest in the first 6 hour period. However, Randall has pointed out that the postoperative excretion is less than that which results from one day of parenteral saline therapy without oral intake. Eliel and associates[2] have shown that the hypochloremic alkalosis, which often is found in patients with intestinal obstruction and prolonged vomiting, and which is so resistant to parenteral saline therapy, usually is correlated with a potassium deficiency and responds quickly to potassium chloride therapy. This also has been emphasized by Elman and associates[4] and by Myers and Kirklin.[11] Eliel and associates,[3] have shown further that metabolic changes were much more pronounced in postoperative patients who were not given potassium than in those who were.

CASE REPORT

A 70 year old white widow was admitted to Denver General Hospital on Sept. 1, 1953, with a gradual progression of gangrene of the left foot for one month. For approximately one year she had been receiving 75 mg. of oral cortisone daily for mild degenerative arthritis of the knees. She had been on no special diet or supplementary medication. She gave no history of diabetes. She was a very obese, elderly, moderately deaf woman who appeared somewhat lethargic but in no acute distress. The temperature was 97.4F., pulse 116, respirations 22, and blood pressure 135/75 mm. of mercury. Significant positive physical findings consisted of a tachycardia with an irregular rhythm, absent arterial pulsation bilaterally below the femoral pulses, and gangrene involving the lower part of the left foot.

Laboratory data on the day of admission showed the hemoglobin to be 12 Gm. per cent and the white count 13,700 per cu mm. with a normal differential count. The urinalysis was normal, except for 2 plus albumin. Blood sugar was 103 mg. per cent, and non protein nitrogen 47 mg. per cent.

The clinical diagnosis was arteriosclerosis obliterans with gangrene of the left foot.

On admission the patient was given a normal diet. Cortisone inadvertently was not continued. A low thigh amputation was scheduled for the morning of her third hospital day. During her second hospital day her blood pressure was recorded on three separate occasions as 110/65, 110/70 and 100/60 mm. Hg. An electrocardiograph was interpreted as showing frequent premature auricular contractions for which she received 0.4 Gm. of quinidine sulfate in two equally divided doses on the night before operation. On the morning of her operation she received demerol 50 mg. and atropine sulfate 0.3 mg. hypodermically at 6:30 as premedication. Upon her arrival in the operating room an intravenous infusion of 5 per cent dextrose in water was started. Eight mg. of pontocaine intrathecally produced a level of anesthesia up to the fifth thoracic spinal segment. Her blood pressure was then recorded as 85/50 mm. Hg. In view of the possibility that her prolonged cortisone therapy during the previous year had resulted in a significant degree of adrenal cortical suppression she was given 75 mg. of cortisone orally. Five mg. of vasoxyl (methaxamine isopropylamine)

intravenously and 15 mg. of vasoxyl intramuscularly produced only a transient rise in her blood pressure. At this point an eosinophile count showed 123 eosinophiles per cu. mm. One mg. of neosynephrine then was given intravenously with a prompt rise in blood pressure to 120/80. The operation proceeded. During the amputation almost constant intravenous infusion of neosynephrine was necessary to maintain the systolic blood pressure above 100 mm. Hg. Blood loss was minimal. At the close of the operation the blood pressure was 94/58 mm. Hg.

Following operation the patient remained lethargic and hypotensive but without tachycardia. At one point the blood pressure fell to 60/40. She was given further cortisone, adrenocortical extract, 500 cc. of whole blood intravenously, and 250 cc. of physiologic sodium chloride solution, all without apparent benefit. Frequent administration of neosynephrine intravenously was necessary to maintain the systolic pressure above 90 mm. Hg. Eosinophile counts two and four hours after operation were 84 and 73 per cu. mm. respectively.

After approximately eight hours of persistent hypotension the possibility of hypokalemia was considered. The electrocardiograph made the day prior to operation was reviewed immediately. The characteristic findings of hypokalemia—in addition to premature auricular contractions—were apparent. A second electrocardiograph again showed evidence of hypokalemia. The patient then was given potassium chloride orally in the form of 1.0 Gm. every three hours in 240 cc. of orange juice. Six hours after the beginning of her potassium therapy her blood pressure had risen to 110/80 mm. Hg and thereafter never was recorded below 100 mm. Hg systolic during her convalescence. Prior to the return of the blood pressure to normal the only sodium the patient received was that contained in the 250 cc. of physiologic sodium chloride solution which immediately followed the blood transfusion to flush the tubing.

The serum potassium level about 10 hours after the start of oral potassium was 2.6 milliequivalents per liter (mEq/L.). At the same time the serum sodium was 123 mEq/L., the serum chloride 91.5 mEq/L., the serum CO_2 combining power 20 millimols per liter (mM/L.) and the non protein nitrogen 28 mg. per cent. The patient was given the regular hospital diet containing 5–10 Gm. of sodium chloride per day with supplementary oral potassium chloride. On the fifth postoperative day repeat laboratory determinations were: serum potassium, 4.6 mEq/L.; serum sodium, 145 mEq/L.; serum chloride, 106 mEq/L.; CO_2 combining power, 18.6 mM/L.; and non protein nitrogen, 47.5 mg. per cent.

The patient was discharged on her thirty-second hospital day asymptomatic and with her amputation stump well healed.

DISCUSSION

This case is an example of operative and postoperative hypotension in a patient with chronic hypokalemia induced by prolonged cortisone therapy and made more profound by a major surgical procedure. The cause of the hypotension apparently was unrelated to anesthesia or blood loss. The possibility of adrenal suppression was considered and cannot be completely excluded. However, the failure of the blood pressure to respond to ACTH, cortisone and adrenal extract makes this unlikely. Electrolyte depletion was of major importance. The sodium deficit probably contributed to her lassitude,[10] but it is unlikely that the small quantity (2.25 Gm.) of sodium chloride given nine hours before the disappearance of hypotension could have been responsible for the result. The best chronologic correlation of improvement with treatment rests with the specific treatment of the patient's potassium deficiency.

If this correlation is valid, hypokalemia should be considered one of the causes

of *unexplained* hypotension, especially when the latter occurs in the operative or immediate postoperative period.

SUMMARY

A case of hypokalemia is described which resulted from prolonged and poorly regulated cortisone therapy and which was exaggerated by the stress of operation. During a major operation hypotension occurred and persisted into the postoperative period. The hypotension disappeared coincident with the administration of potassium chloride. Prolonged cortisone therapy has been re-emphasized as a cause of hypokalemia as well as adrenal suppression.

Hypokalemia may be a cause of unexpected operative and postoperative hypotension.

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MECONIUM PERITONITIS

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Meconium peritonitis is an asceptic, peritoneal inflammatory process caused by a prenatal perforation of the bowel. The escape of meconium from the perforation into the free peritoneal cavity results in an acute inflammatory reaction which later becomes chronic. This chronic peritoneal inflammation is characterized by an obliteration of the free peritoneal cavity with a dense fibrous exudate and the formation of a meconium filled pseudocyst about the site of the perforation. A valuable diagnostic sign is the deposition of calcium flecks in and about the fibrous adhesions.

Secondary infection of the pseudocyst, increased abdominal distention, and respiratory embarrassment supervene within 48 hours after birth in the untreated infant with this disease. Death is inevitable if the perforation is not closed and the continuity of the gastro-intestinal tract is not established.

The cause of the perforation such as meconium ileus, atresia, duplications and malrotations of the intestine are found in approximately 50 per cent of the patients; whereas no cause for the perforation can be found in the remaining 50 per cent. An occasional case has been reported in which the perforation sealed spontaneously and required no treatment.

The diagnosis of this disease should be suspected in any newborn infant with a distended abdomen. It is confirmed by the roentgenologic demonstration of intra-abdominal calcification.

Operation is indicated as soon as the diagnosis is made and is directed toward slow evacuation of the pseudocyst; closure of the perforation, and establishment of gastro-intestinal continuity.

Meconium peritonitis is a rare disease. There are less than 100 reported cases with only 15 survivals, including the patient presented in this report (table I).

Simpson[12] usually has been credited with the first recorded description of meconium peritonitis in a report dated 1834. He presented postmortem findings in 25 new and stillborn infants with evidence of intra-abdominal inflammation. A careful review of his descriptions indicates that only 1 or possibly 2 of the infants could possibly have died from meconium peritonitis.

Agerty and associates[1] reported the case of the first successfully treated patient with meconium peritonitis.

CASE REPORT

The patient is a Negro female infant born at term at Parkland Hospital in Dallas, Texas at 2:20 p.m. on March 17, 1953.

Examination of the infant immediately after birth showed a markedly distended ab-

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domen with a dull percussion note and a questionable fluid wave. The proximal 3 inches of the umbilical cord was distended, measuring 4 cm. in diameter. There was a slow ooze of blood from the cord just distal to its cutaneous junction. For this reason an umbilical clamp was placed on the cord proximal to the bleeding point and very near the abdominal wall. Immediately after the application of this clamp, bile stained fluid began to ooze from the crushed portion of the cord. It was the impression of the surgical staff who saw the patient at that time, that an omphalocele or choledochal cyst was present and that it had been transected by the umbilical cord clamp. Routine blood and urine studies were normal

Three hours after birth the abdomen was explored under open-drop ether anesthesia through a right rectus incision. Upon opening the abdomen, a pseudocyst was entered which contained approximately 150 cc of bile-stained fluid with a sediment which appeared to be meconium. This fluid was removed quickly. The general condition of the patient deteriorated somewhat after this fluid was removed. Further exploration showed that the cyst extended from the costal margins superiorly to the pelvis inferiorly and from one flank to the other laterally (fig. 1).

On the posterior cyst wall, 1 cm. to the right of the midline and on a line 1 cm. superior to the umbilical level, was a rosette of mucosa. The mucosa was everted and two lumens were visible; it gave the appearance of a long established loop ileostomy. There was an area of calcification on the posterior cyst wall above and to the left of the rosette.

Catheterization of the rosette's lumens was done. The left lateral lumen contained some solid meconium, but no obstruction was met for a distance of 10 cm. It was thought that this represented the proximal lumen of an enterostomy. Catheterization of the right lateral lumen showed an obstruction 6 cm. from the rosette. At this point in the exploration the patient's general condition deteriorated further and the operation was terminated quickly in the following manner: a catheter was left in the distal bowel lumen and the rosette was exteriorized.

The infant was maintained satisfactorily for the next 22 hours with intravenous fluids while roentgenologic studies were being made. Following a rectal examination the morning after operation a small mass of very thick tenacious blood-stained mucous passed as the examining finger was removed. Several minutes later, more material passed spontaneously but it was not bloody. Squamous epithelial cells were found microscopically in the material.

A series of roentgenograms made after injection of radiopaque medium into the rectum and into the distal lumen catheter gave the impression that the small bowel was patent as far as the rosette formation, but that an obstruction was present several centimeters distal to the rosette.

Twenty-two hours after the first operation, the infant again was explored by reopening the previous incision. Beginning at the rosette, the bowel, from the rosette to the ascending colon, was separated from the dense fibrous adhesions of the cyst wall. Blood loss from this dissection was alarming. The area of mucosal eversion was found to be a perforation of the ileum 20 cm. from the ileo-cecal valve.

Physiologic sodium chloride solution, tinted with methylene blue, was injected into the distal lumen at the perforation. This passed quickly from the rectum which definitely established the patency of the remaining intestinal tract.

The perforation of the ileum then was closed in two layers and an ileoileostomy was made to by-pass the perforation site. The wound was closed with through and through stainless steel wire sutures.

The infant did well postoperatively. Bowel sounds were present in 36 hours. On the fifth day the patient passed six green mucoid stools. Feedings were commenced on the sixth postoperative day and a normal stool was passed on the eighth day. The wound healed primarily and all sutures were removed on the twelfth postoperative day. The child was discharged from the hospital on the forty-fourth day of life weighing 5 pounds 9 ounces.

At 2 months of age her weight was 6 pounds 9 ounces. At 8 months she weighed 16 pounds; was eating well, and was free from symptoms (fig. 2). The abdominal scar was the only abnormality found on examination.

TABLE I

Survivals following operation for meconium peritonitis

Author	Year	Site of Perforation	Possible Cause of Perforation	Treatment	Remarks
Agerty, Zisserman, and Shollenberger[1]	1943	Lower ileum	Undetermined	Perforation closed	First patient to survive this disease
Neuhauser[9]	1944	Not found	Atresia of jejunum	Side tract entero-enterostomy	—
Brunkow, Nelson, Goodnight, Hunter, and Brunkow[3]	1949	Not determined. Probably in the jejunum about 20 inches from the Ligament of Treitz	Undetermined	Drainage of infected cyst with formation of fecal fistula which closed spontaneously and later on produced obstruction requiring lysis of adhesions	This is probably a case of spontaneous perforation, creation of a fecal fistula by drainage which healed spontaneously and which later on produced intestinal obstruction requiring reoperation
Low, Cooper, and Cosby[8]	1949	Not found	Atresia of distal ileum	Entero-enterostomy by-passing the obstruction	Patient thought to have perforated in utero and healed over at adhesion site
Lee and MacMillan[7]	1950	Cecum	Atresia of splenic flexure of colon	Perforation closed	—
Doyle[5]	1951	Ascending colon	Undetermined	Perforation closed	This perforation apparently took place at or immediately after birth
Doyle	1951	Lower ileum	Undetermined	Perforation closed	—
Packard and Reynolds[11]	1951	Sigmoid colon	Possible trauma at time of birth	Drainage of infected pseudocyst	Patient formed fecal fistula which closed spontaneously
Conrad and Robbins[4]	1951	Upper jejunum	Atresia of jejunum	Bowel resection. Side to side anastomosis	—
Franklin and Hosford[6]	1952	Undetermined	Undetermined	Perforation closed	—

Franklin and Hosford	1952	Undetermined	Undetermined	Exploration and evacuation of cyst, then reoperation and closure of perforation	—
Bendel and Michel[2]	1953	Terminal ileum	Undetermined	Perforation closed. Ileotransverse colostomy. Sigmoid colostomy	Sigmoid colostomy was done as a diagnostic procedure, but actually was not necessary
Nixon[10]	1954	Upper ileum	Volvulus	Volvulus reduced. Side to side anastomosis. Perforation closed	—
Nixon	1954	Mid-ileum	Atresia of ileum	Perforation closed. Side to side ileotransverse colostomy	—
McNeill and Votteler	1954	Terminal ileum	Undetermined	Perforation closed. Ileo-ileostomy	—

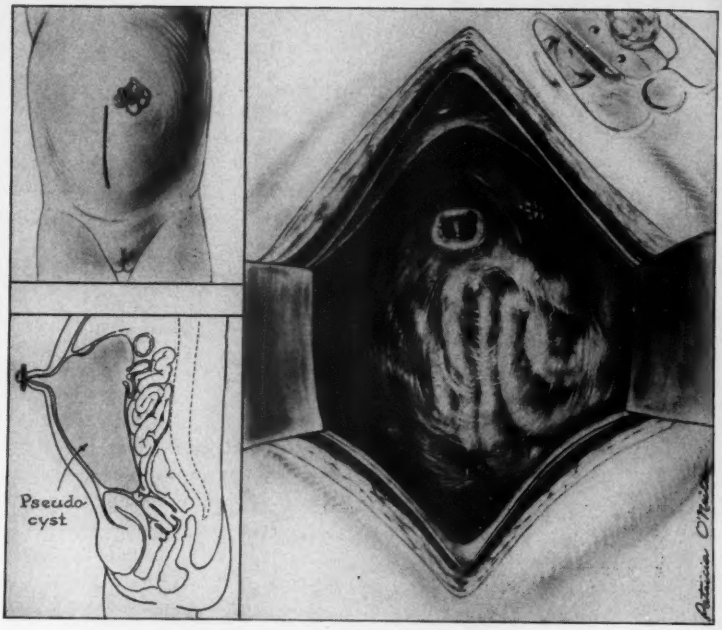

FIG. 1. Artist's sketch of findings at operation

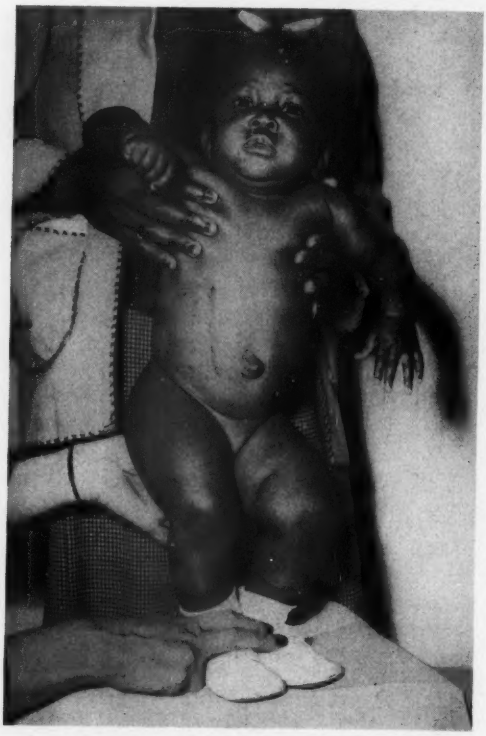

FIG. 2. Photograph of child at 8 months of age

SUMMARY

Meconium peritonitis is a rare disease. Preoperative diagnosis is simple if it is suspected in any newborn infant with a distended abdomen. The diagnosis is confirmed by the presence of intra-abdominal calcification which can be seen by roentgenologic examination of the abdomen.

The important points in treatment of this disease are: (a) immediate laparotomy with slow evacuation of the pseudocyst, (b) location and closure of the perforation of the bowel, (c) establishment of gastro-intestinal continuity with minimal dissection. If there is an obstruction distal to the perforation, it must, of course, be relieved in an appropriate manner.

Acknowledgment: The authors wish to express their appreciation to Dr. Manning Shannon and Dr. Ben Wilson for their helpful advice in the management of this patient, and to the Art Department of The Southwestern Medical School of the University of Texas for the illustration and photograph.

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SPONTANEOUS PNEUMOTHORAX: THE CASE FOR EARLY THORACOTOMY*

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We are discussing true spontaneous pneumothorax and not pleural accumulations of air following trauma, nor occurring as a rather rare complication of tuberculosis. The idiopathic condition typically found in healthy young adults now is known to be unassociated with tuberculosis[16] and many observers have commented on the remarkable lack of association with stress or exertion in most instances. It is obvious that many mild cases are dismissed by the patient or his physician as myositis, neuralgia, or other conditions such as virus pneumonia, and it also is evident that many of the rapidly fatal attacks are misdiagnosed as a heart disease. Blackford[2] found approximately 1 case for every 1,000 students.

At present there still is wide variation in therapy. The patient often is admitted on a medical service for observation or occasional needle aspiration. In the study of 97 consecutive cases of true spontaneous pneumothorax by Rottenberg and Golden,[16] the average time for apparently complete re-expansion was 4 weeks, and the longest 12 weeks. Kircher and Swartel, in their interesting recent analysis,[11] found in 16 patients who were treated with bed rest alone, similar re-expansion curves with an average rate of air absorption of 1.25 per cent per day. They made this the basis for recommendation that patients with less than 20 per cent pneumothorax be treated with bed rest alone and the others by intercostal tube drainage.

Other authors have been impressed with the experience of the thoracic surgeon who always has found the surest way to keep out of trouble in the chest is to expand the lung promptly—to expand it completely—and to keep it expanded. They therefore placed an intercostal drain, which may be done neatly at the bedside through a trocar under local anesthesia, and institute water seal drainage, thus shortening the convalescence to a week[10] or two[11]. There may be little urgency in handling these patients in the peace time armed service hospitals or in the veterans hospitals, but with the private patient who is anxious to return to work in some remote town, time becomes a real consideration. It is unfortunate that we have no conclusive figures in the literature for the rate of recurrence on the same or the opposite side after the various plans of treatment. We wish to recommend that tube drainage be at least the minimal procedure chosen for the hospitalized patient. Hospitalization or close observation is indicated by the mortality rates such as those recorded by Kjaergaard,[12] who reported death in 6 of 200 patients. Tension pneumothorax, always a serious condition, has been found in 10 to 16 per cent of cases. Figures on the recurrence of attacks also vary understandably, up to 45 per cent.[18] Shefts found that of 114 cases 47 were re-

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current or chronic, so if proponents of expectant therapy argue that most patients will re-expand without surgery, they are reminded that this is in itself an insufficient argument. With modern antibiotics and parenteral fluids many cases of appendicitis will subside, yet surgery has long been accepted as the treatment of choice unless definite contraindications such as spreading peritonitis exist.

Pathogenesis of spontaneous pneumothorax remains partially obscure, but seems to involve a probable congenital weakness of the surface of the lung in certain individuals, It has been noted at exploration that the leaks are almost always in connection with blebs or bullae which usually are in the apical region, and that pleural adhesions usually are present. The constant tug on the lung near the leak seems important not only in its creation but also in maintaining it until in the chronic or recurrent case, it epithelializes. Cutler[8] suggested thoracoscopy and pneumonolysis as a lifesaving measure in tension pneumothorax complicating pneumothorax therapy. He stated that the release of tension on the adjacent leak usually permitted it to seal. Brock[6] suggested application of silver nitrate to form pleural adhesions and prevent recurrence, which also was commended by Curti and Poulsen.[7] The production of a chemical pleuritis to prevent recurrence first was conceived by Spengler in 1901. I became interested in this problem while trial was being made of a saturated glucose solution. Great pain was produced, and there was difficulty in posturing the narcotized patient so as to distribute the irritant successively to all pleural areas, as well as difficulty in reaspirating all of the solution rapidly diluted by pleural fluid to permit contact of the inflamed surfaces. Paul, Beattie, and Blades[15] published a report favoring insufflation of iodized talc, finding no significant loss of pulmonary function after several months. We have not followed this policy, not only from fear of late talc granuloma producing eventual fibrothorax, but also because abrasion with universally available dry gauze is so simple, controllable, prompt and uniform in action, and eliminates the foreign body problem entirely.

Enough patients have expanded completely after conservative therapy, only to recur where adhesions were present, to justify our going on record in favor of early thoracotomy, lysis of adhesions, and after conservative local excision of bullae, abrasion of the pleura with dry gauze. Shortly after we settled upon this program an excellent article appeared in *Surgery* by Beardsley and Pahigian[1], reporting this technic in four operations on 3 patients (1 being bilateral, with stages nearly two months apart). They utilized needle aspiration rather than tube drainage after closure, whereas we believe that tube drainage not only is safer in the long run, but reduces excess fibrin deposition with later organization and partial loss of thoracic function. Lindskog,[14] in his recent text on thoracic surgery, stated that there is recurrence in approximately 20 per cent of spontaneous pneumothorax and recommends, "Unless there is some compelling contraindication to major surgery, these patients should be treated by thoracotomy with lysis of all adhesions, and local excision of any visible bullous areas whether or not the site of fistula. The parietal pleura then should be abraded mechanically with dry surgical gauze, and the chest closed with intercostal suction drainage.

The drains are removed when no further air leaks are demonstrated and total 24 hour drainage is less than 50 ml."

Brewer, Dolley, and Evans[4] reported 15 cases of patients with chronic spontaneous pneumothorax in 1950, pointing out that it had been shown repeatedly by bronchospirometry tests that pleural effusion resulting in fibrosis markedly decreases pulmonary function. With surgical treatment rapid expansion of the lung is accomplished with minimal pleuritis and maximum pulmonary function. They were able to find over 100 patients in the literature who were treated by production of pleuritis by introduction of a wide variety of pleural irritants, while in only 31 patients had surgical treatment been applied. In discussing the etiology of the pneumothorax, they mention the presence of intrapleural adhesions and in each of the diagrams of their patients adhesions are shown so located as to place tension on the site of leakage. In all of their patients results were excellent, except 1 in whom decortication was not done. Shefts[17] did thoracotomies in 2 of 61 patients seen during the first attack with recurrence in neither. Twenty-two were treated by bed rest or aspiration, with recurrence in 4, and 37 with catheter aspiration, with recurrence in 3.

Our message therefore is a logical outgrowth of a number of contributions and essentially is that spontaneous pneumothorax, particularly if there is any evidence of recurrence, should no longer be considered a medical disease. The patient should be admitted directly to the surgical service, even though thoracotomy may not be done, just as a patient diagnosed as having appendicitis is handled, although laparotomy is not always done. It is our contention that enough now is known of the incidence, pathogenesis, and prognosis, and that thoracotomy under proper conditions has attained such great safety, particularly in these typically healthy individuals, that we are no longer justified in the majority of patients to use conservative treatment with its attendant ultimate risk and immediate economic loss.

Our own series of 12 patients who were treated by thoracotomy consistently supports the recommendations of Brewer, Beardsley, Lindskog, and others, although papers continue to appear suggesting drainage alone. For example, Briggs, Walters and Byron[5] reported last year on 84 cases of spontaneous pneumothorax, including three deaths in untreated patients proved by autopsy to have had tension pneumothorax. They found the history of recurrence in 17 per cent. Forty-five patients were treated with bed rest alone. They found that complete re-expansion required about 34 days by that method, whereas an average of 22 days were consumed by needle aspiration, and the lung usually re-expanded in three days after use of catheter and water-sealed drainage, permitting early discharge from the hospital.

However, these reports still fail to consider the occasional late recurrence following failure to cause pleural symphysis, with an occasional empyema or fatal tension pneumothorax as well as the more common chronic pneumothorax with marked function loss. Moreover, no mention is made that conservative resection of bullae prevents their growth at the expense of sound lung. Thoracic surgeons have come to realize that resection of pulmonary cysts always should be conserva-

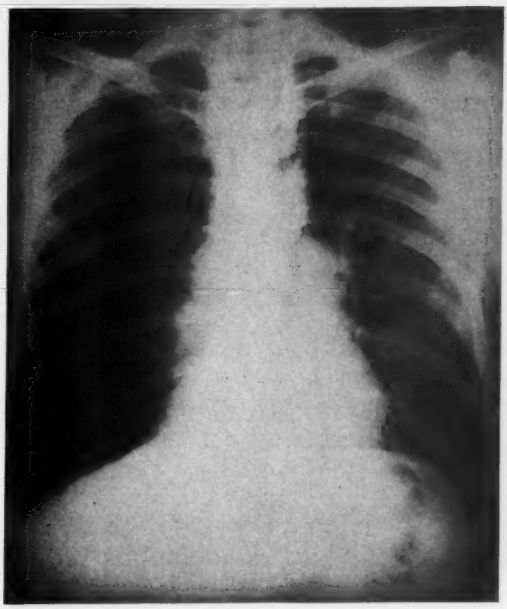

FIG. 1

tive and local where possible, rather than by removal of whole lobes or even segments of lung since cysts may later appear in other areas. By resecting superficial cysts and permitting relatively sound lung to adhere to the chest wall, we believe that further dissection by these bullae or cysts at least is minimized.

The nature of the adhesions produced by the dry gauze technic should be mentioned here. The technic is not new, having been described by Lilienthal in his text book on thoracic surgery[13] published in 1926. It was used to insure adherence of a sound lobe before resection of an adjacent lobe for suppuration in anticipation of bronchial stump leakage and in order to localize resultant empyemas. On re-opening a chest after dry gauze symphysis the lung appears to glide with respiration, and yet is found to be uniformly sufficiently adherent, if some care was used in applying the gauze evenly, to eliminate potential pleural spaces.

Two cases are presented to illustrate our philosophy and our technic. The first is that of a young engineer, an outstandingly healthy ex-fighter pilot who had sudden onset of dyspnea and right chest pain while waiting in line at an airport terminal. Being of stoical disposition, he proceeded to embark and as the plane gained altitude found himself in agony from dyspnea. He was somewhat improved at the first landing, but again was in extremis as the plane ascended. He therefore deplaned and reported to our Clinic for treatment. His initial film showed almost complete collapse of the right lung (fig. 1) and I suggested to the internist in charge that thoracotomy be done. He objected that no adhesion had been

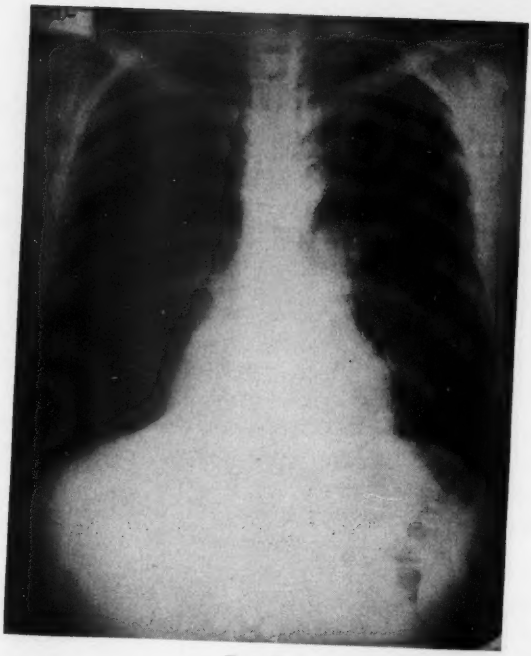

FIG. 2

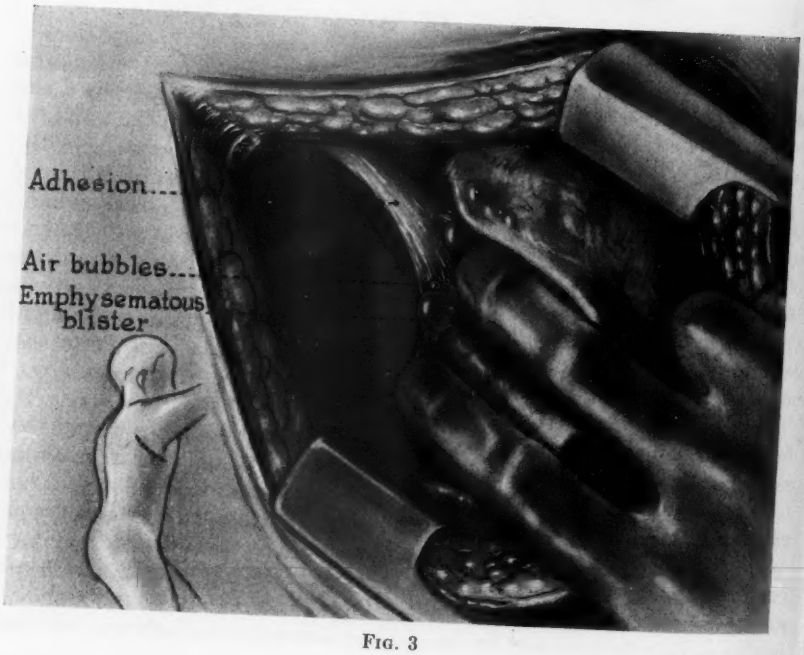

FIG. 3

demonstrated and withdrew a liter of air by needle. The following day a roentgenogram (fig. 2) again showed the lung almost completely collapsed, but fortunately enough air lay between the apex of the lung and the adjacent mediastinum to demonstrate an apical adhesion. We then agreed to leave the decision to operate to the patient, informing him that in many good hospitals he would be treated merely by tube drainage, but that we believed we could not reasonably guarantee against recurrence without thoracotomy, after which he could be released in eight days as cured. He explained that his mission was to supervise heavy construction deep in Old Mexico, and that he wished to take no chances with recurrence, and in fact would accept operation to minimize hospitalization. At operation a typically well-localized area of bleb formation was found tented by an adhesion and a leak readily demonstrated (fig. 3). After dividing the adhesion and over-sewing the bleb, the pleura was rubbed and the lung re-expanded. He was discharged on the sixth day and sutures were removed on the eighth day. He has remained symptom free for over two years.

Another case is that of an Air Force pilot, grounded for recurrent pneumothorax. His initial attack, while playing cards, occurred in 1949, and a con-

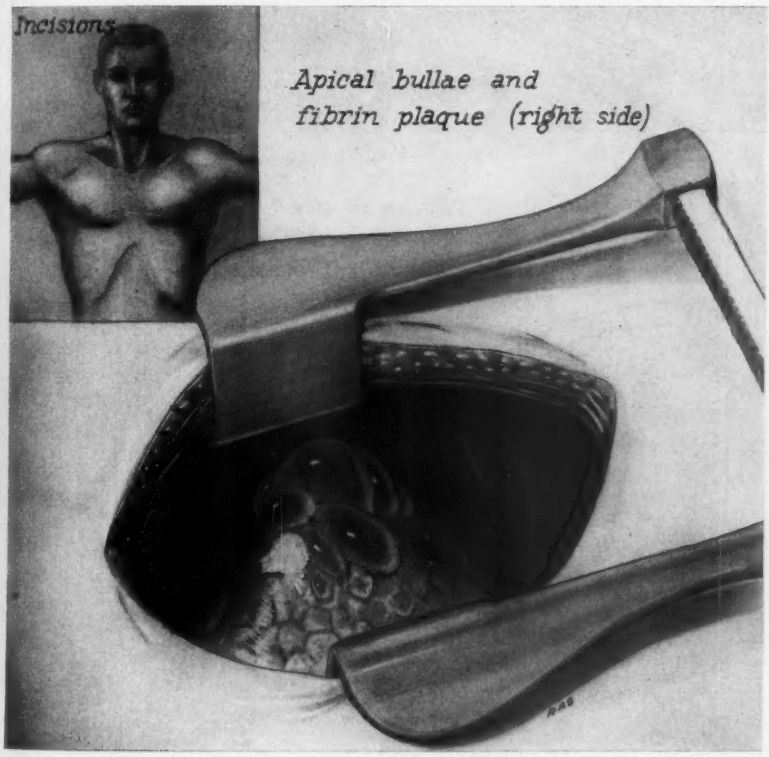

FIG. 4

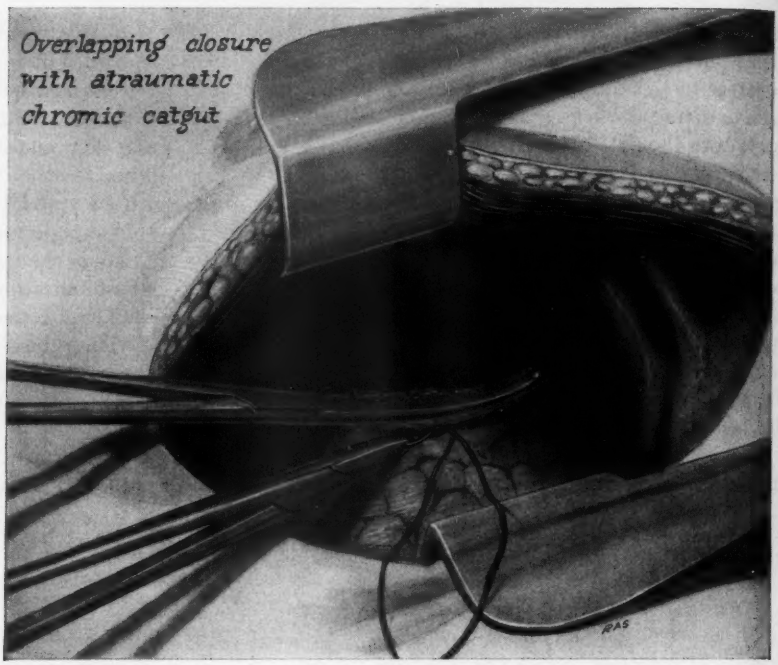
Overlapping closure
with atraumatic
chromic catgut

FIG. 5

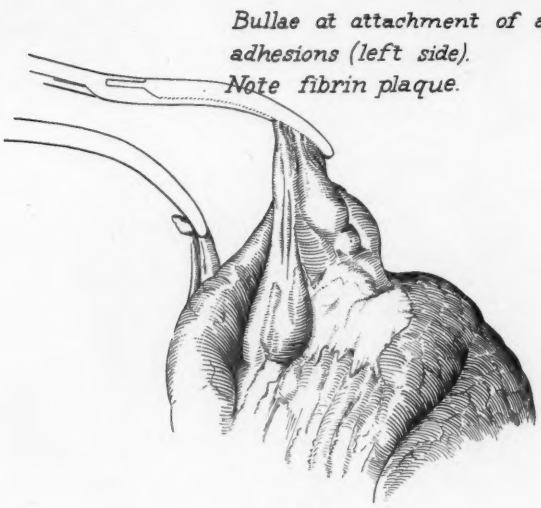
Bullae at attachment of apical
adhesions (left side).
Note fibrin plaque.

FIG. 6


Ligation of blebs after
emptying by torsion

FIG. 7

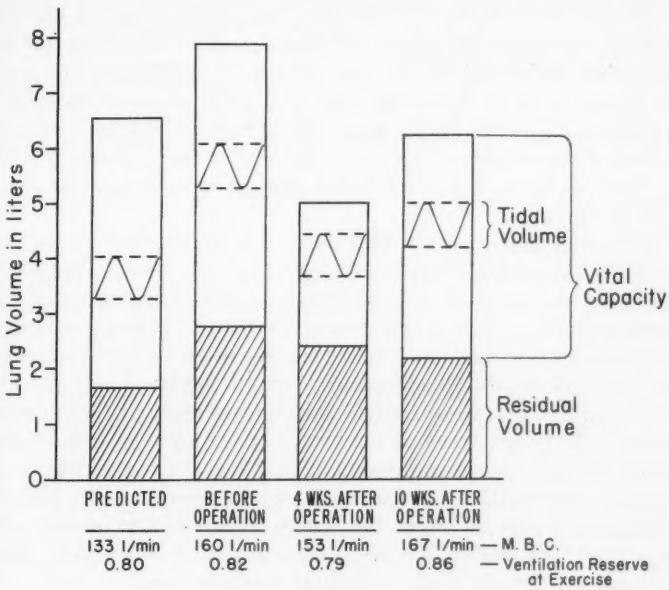
Lung Volume in liters
0
1
2
3
4
5
6
7
8
Tidal Volume
Vital Capacity
Residual Volume
PREDICTED
BEFORE OPERATION
4 WKS. AFTER OPERATION
10 WKS. AFTER OPERATION
133 l/min
0.80
160 l/min
0.82
153 l/min
0.79
167 l/min — M. B. C.
0.86 — Ventilation Reserve at Exercise

CHART I

tralateral attack the following year was followed by recurrence on the original side four months later. At the time he reported for treatment, he had had no frank pneumothorax for three years, but still was grounded because of suggestive cystic changes in the lung apex. After thorough examination of the patient, including pulmonary function studies, in which air trapping was demonstrated by use of the nitrogen meter,[3] bilateral thoracotomy was done through short inframammary incisions (figs. 4 to 7). It is believed that a conservative resection of the cystic areas in both apices of this patient will make a marked difference in his future prognosis. After his discharge on the tenth postoperative day arrangements were made for serial function studies to demonstrate the minimal loss of function by this technic. The second study made only three weeks after discharge from the hospital, showed a remarkable absence of change in the maximum breathing capacity (chart 1).

CONCLUSIONS

In conclusion, we present our own contribution, the concept that thoracotomy need not be withheld in an initial attack, if certain criteria are met. Previous authors have stressed thoracotomy in the patient whose condition is chronic, or has recurred at least once.

We would not be put in the position of lightly recommending the opening of any body cavity, but if a patient has access to suitable facilities, and if a surgeon with experience in thoracic problems is available, it does seem no longer justifiable to adhere to an arbitrary rule of surgery only for recurrences. We owe it to our patients to recognize that the situation has changed with our present mastery of the open chest and its subsequent care. The availability of modern antibiotics might be mentioned as an additional safeguard, although these problems do not involve infection. The fact that 9 of our 12 patients who were operated upon and nearly half the number of the series studied by Shefts, became chronic or were repeaters, is to us strong indication that previously reported recurrence figures are misleading.

If the patient has more than a trifling collapse, if there are adhesions (which we found in all of our patients but 1, although often difficult to demonstrate preoperatively), if the patient is in good health (as these patients usually are), if the patient expects to work or travel away from ready access to a hospital (as most do in this restless age), if there is hemopneumothorax, (for practical purposes always involving a torn adhesion and with a mortality rate as high as 20 per cent—a condition for which early thoracotomy already has been described[9]), and finally if the proper facilities mentioned above are available, it seems regrettable to offer a prolonged and less than certain cure by aspiration or even by tube drainage, since neither can remove the pathology we usually find present. Even if pleural irritants are introduced in such a manner as to reliably obliterate all areas without undue late fibrosis—a difficult problem—we have admittedly left cystic areas to expand at the expense of adjacent lung.

We are aware that a study of several dozen cases, followed for at least 10 years after operation, will be required for ideal quantitative proof of our thesis, but

the method is presented at this time in the hope that others will be sufficiently impressed by the basis provided for its trial, to offer it to suitable patients with the knowledge that there has been uniformly successful precedent in our series of patients treated by thoracotomy and followed for periods up to four years.

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EPISODIC COMA DUE TO MEAT INTOXICATION AS A FATAL COMPLICATION OF PORTACAVAL SHUNT IN THE HUMAN BEING

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INTRODUCTION

Meat intoxication is a syndrome originally described in the dog having a portacaval shunt and fed on large quantities of meat. It is characterized by muscular rigidity, ataxia, loss of function of the special senses, (particularly of vision), stupor, coma, and death. The neurologic state can be produced in most animals, but forced feedings with meat may be required. Withdrawal of meat, if done early enough after the signs appear, causes the animal to revert to normal.

This phenomenon has been reproduced and reported by many observers.[2, 8, 10] A report on a similar condition in man recently was made by McDermott and Adams[5] whose patient developed episodic stupor after a portacaval shunt operation. These authors were able to demonstrate the association of elevated blood ammonia levels with the clinical state of disturbed consciousness. A sharp rise in blood ammonia levels accompanied the onset of the symptoms during every episode. They postulate that ammonia is the substance which interferes with the normal metabolism of the central nervous system and impairs its function.

Recently we had the opportunity to observe a patient with a portacaval shunt who exhibited, over a period of 18 months, episodes of varying degrees of clouding of consciousness and confusion. The experience gained from the clinical observation of this patient seems to justify certain conclusions which should be helpful in the management of patients who suffer from this alarming although remediable complication.

CASE REPORT

F. M., a 48 year old married Filipino hotel clerk was admitted for the first time to the Seattle Veterans Administration Hospital in December 1951. He gave a history of having had a sudden hematemesis in March 1951, for which he had been hospitalized at the U. S. Public Health Hospital in Seattle. At that hospitalization he was found to have esophageal varices and poor liver function. He had declined surgery. He had had another bout of hematemesis which prompted his admission in December.

On examination he was a slender man of dark complexion, without icterus. He weighed 57 kilograms. The blood pressure was 116/78, the pulse 68, and the temperature 97F. The head, neck, chest and lungs were normal. The heart was slightly enlarged to the left. A nontransmitted grade II systolic murmur was heard in the second left interspace. The abdomen and genitalia were normal. External hemorrhoids were present. The extremities did not show edema and only a few small varices.

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Reviewed by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions of the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Esophageal varices were seen again on esophagoscopy. Detailed laboratory studies showed only fair liver function at this time. (See summary of laboratory data in table I).

Early in January 1952 a side to side portacaval anastomosis was made, leaving the portal vein intact. The pressure in the portal system was reduced from 300 to 130 mm. of saline by the operation. The liver was found to be small and cirrhotic. A biopsy showed periportal fibrosis with rather marked round-cell infiltration (fig. 1). The postoperative course was benign. He went home after a month of leisurely convalescence.

For the next seven months he did well, returning to his work as a clerk. There was one episode of swelling of the legs, which was relieved by compression bandages. In August 1952 the swelling recurred, together with swelling of the hands and face. He noted a *tightness* in the jaws with difficulty in opening the mouth. His appetite had been excellent and the diet unrestricted. The edema was attributed to be due to a low albumen, and resolved on a high protein, low sodium diet.

One month later the patient re-entered the hospital with epigastric pain and nausea. A duodenal ulcer was demonstrated and treated by frequent milk-cream feedings, Banthine, and phenobarbital. He became confused; developed urinary retention, and weakness of his hands. The symptoms were thought to be due to Banthine toxicity because they disappeared with the discontinuance of the drug. However, the drug was started three days later without any further symptoms. The milk and cream was not given after the onset of the confusion. The edema diminished, but was still a problem.

There were two more hospitalizations for abdominal pain attributable to the duodenal ulcer. Prior to the admission in early September 1952, the patient became *nervous* at home, with tremor, drooling, mental confusion, and urinary frequency. He had no such symptoms in the hospital and received no specific medication.

In April 1953, 15 months after the portacaval shunt had been done, he re-entered the hospital after five days of severe abdominal pain which was treated with anti-acids and milk. He was lethargic, vague, slow to respond to questions, but oriented. No blood loss was evident. The pain resolved after frequent feedings of milk fortified by meritene.* After five days on this regime he complained of blurred vision, and wandered about the ward at night. He gradually developed thick speech, confusion, disorientation, ataxia and loss of all inhibitions. All medication (which included atropine and Banthine) was discontinued. Within two days the patient was back to his normal sensorium. He was placed on a regular diet (one half of the milk and protein formula) and Banthine. On this regime he remained well and was discharged.

The ulcer pain recurred at home. He vomited occasionally—once the vomitus was *coffee grounds* in character. An episode of continuous vomiting for four days necessitated rehospitalization in late July 1953. He improved on milk and cream with antispasmodics, and was transferred to a nursing home.

One week later his wife returned the patient by ambulance to the hospital stating that he vomited constantly; was *blind*, had to be led about, and finally was confined to bed. His speech was scarcely intelligible, and he was described in the record as ataxic, weak, and *deteriorated*. He complained that he could not see. No history of abnormal dietary intake was obtained. Neurologically there was constriction of the visual fields; lack of coordination, slowness of action and easy fatigability. The following day these signs disappeared and he remained lucid until the tenth day. On the tenth day in the morning he complained of *smarting* of the eyes, stiffness of the jaws, and leg pains. He was confused, wandered about aimlessly and was disoriented. He progressed into a deepening stupor which persisted with restless periods for 10 days. Prior to these symptoms he had been getting milk and cream, 30 cc. every two hours, but no other medication. Arsenic poisoning was suspected, and he was given BAL in oil, with supporting parenteral fluids. All protein intake was discontinued for three days. On the fourteenth day there was spectacular recovery from the coma. He made frequent comments upon his restored vision. For the next two weeks he remained well, re-

* Protein supplement by Mead Johnson.

TABLE I
TABULATION OF LABORATORY DATA GATHERED DURING HOSPITAL ADMISSIONS

Shaded Dates Signify Coma Periods		1951	1952									1953																						1954								
		DEC	JAN				FEB			MAR	APR	AUG		SEP	OCT	JAN		APR			JUL		AUG		SEP			OCT		NOV		DEC	JAN		FEB					
		20	28	7	21	24	28	1	14	4	1	1	26	14	14	14	16	2	3	10	22	25	27	14	30	7	15	18	12	26	19	23	24	1	7	18	11	15
Blood Chemistry	Protein Total	6.4			6.4				7.1		6.3	6.7	6.3		6.8	6.6		5.8	6.0							4.5			6.2	5.2		6.5			6.1							
	Albumin	3.5			3.6				3.2		2.3	2.4	2.0		2.2	2.6		2.4	2.3							1.5			2.0	1.9		2.0			2.0							
	Globulin	2.9			2.8				3.9		4.2	4.3	4.3		4.6	4.0		3.4	3.7							3.1			4.2	3.3		4.5			4.1							
	B.S.P. Retention	trace ret	14%						25%		26%				37%	37%								46%	47%																	
	Ceph. Flec.		3+	3+						3+	4+						3+																								
	Thymol T		1.8	3.3								7.1						3.4	3.5		6.0	10.1		6.0						3.4	3.6				2.6							
	Alkill Phosphate			7.1	16.3													9.7	14.0	15.7	12.3	6.1								21.8	7.1											
	Bilirubin		1.2					3.0		1.5			2.4				2.5	1.9	2.1		1.7	2.6		1.9				1.7	4.4	3.6				2.0							
	Van den B Indirect			0.3							0.8	3.2																													
	Van den B direct			0.8							1.4	0.6																													
	Icteric Index								28	10	12																														
	Prothrombin Time	84%		80%					45%		51%	37%		42%				54%	39%			30%		29%	32%	32%			37%			39%									
	Coagulation Time	5'					3'33"				2'5"																														
	Bleeding Time	2'					2'40"				1'15"																2'														
	Blood Sugar																			84		180	82		126	77		77			145		86		87				108	
Renal Function	Urine Spec. Gr.	1.013										1.015	1.011			1.020		1.013				1.008	1.005	1.011		1.010										1.011				
	Reaction pH	Alk										Alk	Alk			5.8		5.5				6.5	6.5	6.0		7.0										7.5				
	Sediment	Occ Wbc										Wbc Rbc	Wbc			2 Nbc		0					Wbc	0		0										0				
	Urea Nitrogen										13	13		15				15			15	11			8						12	12								
	Creatinine											1.1																		1.8										
	Serum Sodium MEq.													143						140		Cl 108			Cl 103					No 150 Cl 111		145 102			150 119				
	Serum K MEq.													3.9						4.3										3.5		4.3			4.0				
	Serum CO_2 MEq.																			25		28		25	27					25		27			27				

		1951	1952															1953																1954						
		DEC	JAN									FEB	APR	JUL	AUG	SEP				OCT	JAN	APR		JUL		AUG		SEP	OCT	NOV			DEC	JAN	FEB				
Shaded Dates Signify Coma Periods		19	20	21	30	7	17	21	24	25	28	30	11	1	31	26	9	14	16	20	14	14	2	10	25	27	14	30	15	2	12	16	19	23	1	18	11	15		
Hematology	Blood Hb	7.0	9.8	12.4	12.8				9.8		8.9	12.8	14.6	12.8	13.9	13.3	13.3	13.3	12.5	13.3	13.9	13.6	15.2					13.6	12.8	13.2							11.8	13.1		
	Blood Ht		32%	39%	36%		33%		32%			41%	37%	39%	40%	38%	36%	37%	35%	38%	39%	40%	37%		40%	35%		30%	35%	36%						40%	33%			
	Total WBC	3100	3100		3900		3700							3500	4300	5400			4400	4500	4000	6500	5500		4300	6000		7000	5350	7900						7600	5350	8000		
	Serum Amylase															123							165	174																
Gastric Analysis	Stool Guiac			NEG	NEG	NEG	4+	Trace	4+	Trace			NEG	NEG		NEG						NEG		NEG		NEG	NEG	Trace		NEG		NEG							
	Volume					650																			145														
	Free HCL					0°																			38°														
	Total HCL					8°																			62°														
Spinal Fluid	Pressure																	NOR.		NOR.																			
	Kolmer																	NEG		NEG							NEG												
	Protein Mgm %																	14		10							13												
	Cells																										1 Cell											
	Urine Porphyrin																														NEG	NEG	++	++	NEG	+			
	Urine Coporphyrin																													+									
	Weight in Lbs		124		127								127.8	140												142.8	139												

Shaded Dates Signify Coma Periods

Urine Albumen & Sugar Negative Throughout Course

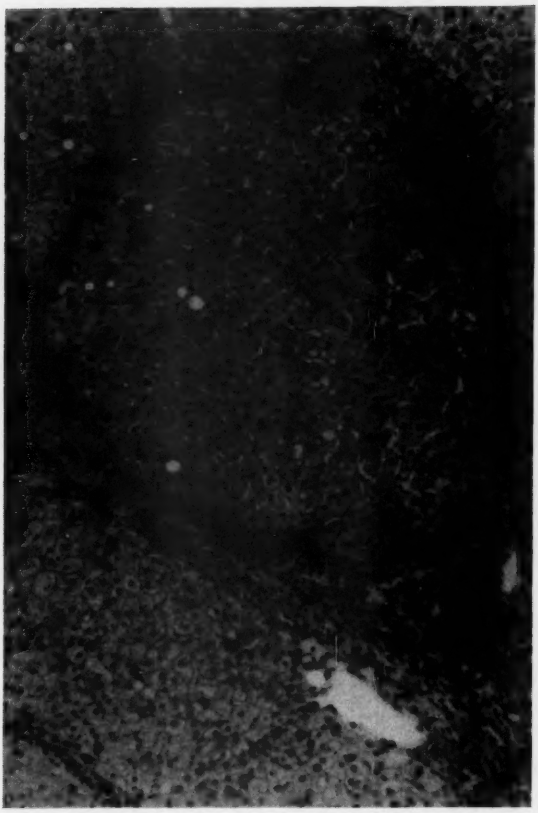

FIG. 1. Photomicrograph of the liver biopsy specimen taken at the time of the portacaval shunt shows striking perilobular round-cell infiltration with some periportal fibrosis. The hepatic parenchyma apparently is regenerating in spots, and in others fatty infiltration is present. The surgery was done during an active phase of liver change (H & E × 100).

ceiving as his only medication 1 cc. of BAL in oil intramuscularly daily. No ulcer therapy was given.

He went home to return again to the hospital in late September 1953 with a recurrence of the stinging in the eyes, blindness, emotional disturbances, disorientation and confusion progressing to stupor and coma. No unusual food or drug intake was noted. The coma would lighten enough for him to take milk and cream and vitamins, then it would deepen. This cycle continued for six days and then cleared, after the depth of coma caused a switch to intravenous glucose feedings. Again the patient commented that he could *see again*. This pattern recurred repeatedly. It was uniformly reproduced on each occasion, although sometimes not going through the entire cycle.

The initial complaint of the patient when going into one of his attacks was that of a gritty feeling in his eyes, or photophobia and soreness of eye movements, blindness, difficulty in moving his jaws, and generalized weakness. At this stage he showed ataxia, dysarthria, confusion and clouding of consciousness, but would respond to questions. He exhibited puerilistic, regressed behavior. This state then either reverted to normal or proceeded into a deep

TABLE II
Diagramatic Representation of The Clinical Course of The Patient Showing The Frequency of

TABLE II

Diagramatic Representation of The Clinical Course of The Patient Showing The Frequency of The Episodic Coma

stupor. During the stuporous periods he no longer responded to verbal or painful stimuli; thrashed around restlessly; smacked his lips; uttered monotonous sounds like *ah* or *uh*, and was incontinent.

On examination he would lie on his bed with dilated pupils which showed varying degrees of reaction to light; he showed snout reflexes; paratonic rigidity on attempts at passive movements; active deep tendon reflexes, but no pathologic reflexes. The temperature, blood pressure, pulse and respirations always remained within normal limits.

The cerebrospinal fluid remained chemically and physically normal at all times. Electroencephalograms were obtained on several occasions. While, during the lucid interval, the record showed only minor abnormalities in the sense of moderate voltage, diffuse intermittent theta waves, the tracings taken during the periods of delirium exhibited such severe abnormalities as diffuse almost constant high voltage theta and delta waves, characteristic of states of disturbed awareness.[9]

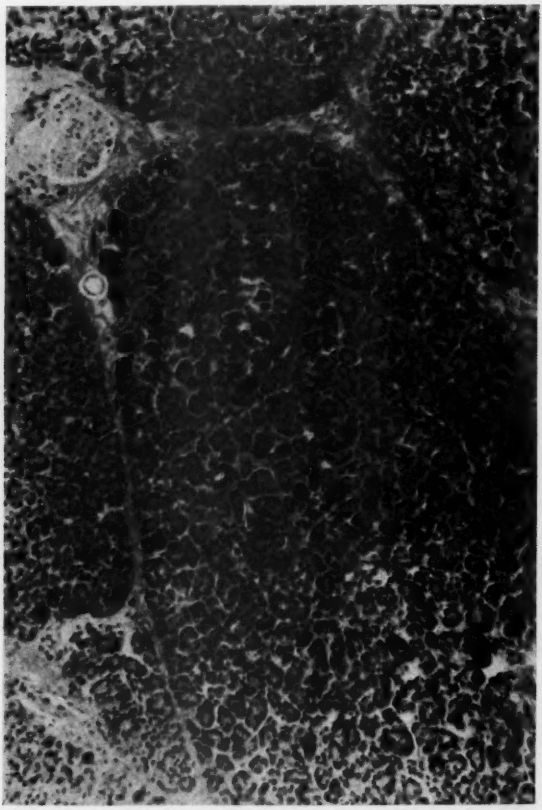

FIG. 2. This photomicrograph was taken of a section of the liver obtained at autopsy two years after the portacaval shunt (and the section in figure 1). There is no inflammatory reaction; however, the periportal and perilobular fibrosis is striking. The liver cells appear to be in surprisingly good condition, although there is nuclear pyknosis, and some fatty infiltration (H & E × 100).

The final episode occurred after a restless night on Feb. 19, 1954. He had been lucid and worked in occupational therapy the day before. There had been some increase in the epigastric pain, but no milk and cream had been taken. He was found in stupor which deepened into coma. There was no emesis or diarrhea. The pulse remained about 80. Respirations were labored.

He died after three days in coma without change in vital signs, or icterus. (The clinical course is presented diagrammatically in table II.)

Autopsy revealed a small, grossly cirrhotic liver, with more fibrosis than noted at operation two years previously, but less inflammatory reaction (fig. 2). There was a large duodenal ulcer penetrating into the liver bed. A large quantity of blood was present in the small bowel and colon; however, the rectum and sigmoid contained normal stool. The side to side portacaval anastomosis was widely patent (fig. 3). Some varices were present in the esopha-

FIG. 3. The dorsal view of the opened inferior vena cava shows the patent portacaval fistula measuring 1 centimeter in diameter. This was a side to side portacaval shunt. The terminal esophagus has been turned to lie adjacent and to the left of the vena cava. The persistent varices with intact epithelium are visible. The shunt had been functioning two years when the patient died in coma following bleeding from a duodenal ulcer.

gus, but there was no ulceration. Sections through the duodenal ulceration showed a definite vessel in the center containing a thrombus.

DISCUSSION

The illness of our patient was characterized by intermittent episodes of delirium with varying degrees of disturbance of consciousness. These attacks began after the portacaval shunt had been done to relieve the portal hypertension on the basis of moderately severe cirrhosis. The attacks were at first one to two months apart, of short duration, and associated with only minor degrees of clouding of consciousness. As time went by the attacks became more frequent; lasted longer and were characterized by deep stupor or coma. During this period no major disturbance of electrolytes or acid-base balance was observed.[4] He remained in good general physical condition despite the evidence of progressive chronic hepatic failure. He did not develop any permanent neurologic deficits. No abnormality of pulse, temperature, blood pressure or respiration was noted. Interval electroencephalographs showed only very minor abnormalities in the sense of diffuse moderate voltage and intermittent theta activity.

This clinical course does not fit the usual picture of hepatic coma. Hepatic coma usually is of shorter duration and does not show the intermittency of our patient's episodes.[6] It is assumed that the portacaval shunt changed the normal course of hepatic failure to the extent that intermittent morbidity, which ordinarily accounts for 50 per cent of the mortality of patients with hepatic insufficiency, is greatly decreased and therefore the life expectancy is prolonged beyond the previously accepted limit. (This is variously quoted as 3 to 12 months after ascites appear or 1 to 3 years after the first evidence of cirrhosis.[1] The length of life in this patient would allow liver function to deteriorate to the extent that exposure to various drugs or even the products of intermediary metabolism, ordinarily detoxified in the liver, would result in periodic intoxication. In favor of this point of view is the relatively long interval between the portacaval shunt, the occurrence of the first attack (six months), and the more and more frequent and increasingly long and severe episodes.

Patients with marginal liver function and dogs with Eck fistulas are known to be particularly sensitive to toxic agents.[6] (The possible exception is the dog with a side to side portacaval shunt with the portal vein intact.[3] Therefore, many different drugs were suspected in our patient, over a period of time. These included Banthine, phenobarbital, arsenic, atropine and paraldehyde. However, close observation showed that, on the one hand, a certain drug would not induce the toxic state regularly, but rather would be well tolerated for long periods of time, and, on the other hand, that the episodes occurred at times when all medication had been discontinued. Investigation of the possibility of periodic exposure to some other toxic substance did not disclose such an exogenous poison to be present. Moreover, the occurrence of the attacks was repeatedly related to the intake of large doses of milk and cream fortified by protein supplements.

It was striking that all of the episodes were exactly alike. It would be unusual, if different toxic substances were responsible, that they would not present cer-

tain specific aspects. This, too, suggests the activity of an endogenous toxin. The pattern of the episodes showed apparent spontaneous recovery after the patient became unable to eat a regular diet due to various degrees of loss of consciousness, and substitution for the meals of intravenous carbohydrate feedings. Such self-limiting mechanisms well could explain the course if constituents of the diet or products of intermediary metabolism were involved. This could be anything aside from carbohydrate.

With the knowledge of animal experimental work with meat intoxication[2, 3, 4, 8, 10] and the case reported by McDermott and Adams,[5] one may conclude that accumulation of a product of protein metabolism, notably ammonia, was the noxious substance in our patient. It is, however, conceivable that other products also could play an important role.[11] The similarity between the stuporous episodes of our patient and the episodes noted by others in the meat intoxicated dog is striking. The sequence of lachrymation, blindness, ataxia, stupor, and coma of recurrent character constitutes a true syndrome in man and beast.[7] It is even conceivable that the intolerance these patients show to gastrointestinal bleeding (our patient died of a relatively mild hemorrhage) represents intoxication by the absorption of the protein of the blood in the intestinal tract.

In view of these considerations certain pertinent conclusions are suggested, which may be of practical importance to those treating portal hypertension in cirrhotics. The patient with a cirrhotic liver is by nature unable to handle certain substances which are ordinarily nontoxic. The addition of a by-pass for portal blood renders the patient with cirrhosis even more susceptible to toxic effects of drugs and products of intermediary metabolism. As the cirrhosis runs its natural course, the liver fails gradually in its role as a detoxifier until death comes from some noxious substance which would scarcely affect the normal person. In the treatment of cirrhosis, death from the complications of portal hypertension can be prevented or postponed by shunting procedures, but in so doing metabolic dangers are introduced. Some of these may be avoidable, and an increasing awareness of the nature of these substances will serve to prolong the lives of these patients.

From this study it is apparent that patients with portacaval shunts should receive a high carbohydrate, low fat, low protein diet and that the administration of drugs, particularly sedatives which are detoxified in the liver, should be avoided.

SUMMARY

The history of a patient is reported who, following a portacaval shunt for portal hypertension secondary to cirrhosis, developed over a period of 18 months episodes of varying degrees of disturbance of consciousness strikingly similar to those described in Eck fistula dogs after *meat intoxication*. These episodes were characterized by a gritty feeling in the eyes, blindness, ataxia, confusion, stupor and finally coma. They occurred with increasing frequency and ultimately caused the death of the patient. The specific etiology of the episodic coma was not determined; however, it appeared to be related to the protein intake and terminally

to a gastro-intestinal hemorrhage. The episodes were quite different from classical hepatic coma.

It is postulated that the episodic coma was due to some product of the intermediary protein metabolism which the failing liver could neither receive (because of the portacaval shunt) nor adequately detoxify (because of its disease).

A patient with cirrhotic liver and a portacaval shunt is therefore in danger, not only from the well known drugs ordinarily detoxified by the liver, but also from his diet and the products of intermediary protein metabolism.

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HEMANGIOMA OF THE GASTROINTESTINAL TRACT WITH CASE REPORT OF A POLYPOID CAVERNOUS HEMANGIOMA OF THE JEJUNUM

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Hemangioma of the gastrointestinal tract is a rare lesion. The esophagus and stomach seldom are involved; most cases occur in the small and large bowel. The condition should be considered in the differential diagnosis of intestinal bleeding with or without signs of intestinal obstruction.

CASE REPORT

F. T. D., aged 25, a white man, first was admitted to the Veterans Administration Hospital on Feb. 6, 1952 complaining of weakness. He stated that he had been in good health until three or four weeks before admission at which time he developed weakness which had been progressive. He had had no disturbance of bowel function and he had not noticed any blood in his stools. He had had no indigestion and no weight loss. There was no history of any bleeding tendency in his family history, and his past history of minor illnesses was not related to his gastrointestinal tract.

Physical examination: He was extremely pale and somewhat apathetic; his nail beds and mucous membranes were pale and there was a yellow tint to the skin. He was 6 feet tall and weighed 162 pounds. His blood pressure was 96/70. The remainder of the physical examination was within normal limits.

Laboratory examination: The positive findings were a hemoglobin value of 5.2 Gm. per 100 cc., a hematocrit of 19 per cent, a red blood cell count of 3,050,000 per cu. mm., and platelets 308,000 per cu. mm. The coagulation time was 11.5 minutes (Lee & White), bleeding time 2.5 minutes, and prothrombin time 80 per cent of normal. The red blood cells showed anisocytosis, poikilocytosis and hypochromia, and reticulocytes 1.2 per cent. The white blood cell count was 9,400 per cu. mm. A urinalysis and serology studies were within normal limits. The serum bilirubin was 0.4 mg. per cent, and the icterus index was 18 units. The Van den Bergh test gave a prompt direct reaction. A gastric analysis done two days after admission using a standard alcohol test meal, showed 104° total acidity and 80° free hydrochloric acid. Four stool examinations showed occult blood. A bone marrow aspiration was reported as "Bone marrow exhibiting hyperplasia of the erythroid elements". Roentgenologic examinations of the chest, stomach, esophagus, duodenum and colon were reported normal.

Therapy consisted of general care and the administration of 2000 cc. of whole blood. His hemoglobin rose to 11 Gm. per 100 cc., and the red blood cells to 4,350,000 per cu. mm.

He was discharged on May 12, 1952, with instructions to return in three months for further observation. He was not seen again until May 7, 1954 when he was readmitted complaining of weakness, occasional abdominal cramping and intermittent passage of tarry stools.

Since his previous period of hospitalization he had served a short period of time in the Army which was terminated after a 60 day period of hospitalization because of weakness

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and anemia. Recently he had been employed as a clerk in an aircraft factory. He had taken iron by mouth to combat his weakness and anemia. He had been hospitalized elsewhere in a medical center for approximately two weeks before admission to the Veterans Hospital. During that time extensive studies and examinations had been made. No definite diagnosis had been made except severe anemia due to blood loss.

Examination on readmission showed essentially the same findings as on the previous admission except that the blood pressure was 110/60 and enlarged discrete lymph nodes were found in the groins and axillas.

Laboratory tests showed a hemoglobin of 7.9 Gm. per 100 cc., a red blood cell count of 2,940,000 per cu. mm., and a mematocrit of 29 per cent. The red blood cells showed anisocytosis, poikilocytosis, and hypochromia. There was 1 plus albumin and a few pus cells in the urine. The Kahn test was normal. The serum bilirubin was 0.8 mg. per cent, the thymol turbidity showed 2 Maclagan units, the cephalin flocculation test was negative at 24 and 48 hours, and the icterus index was 13 units. The gastric analysis with an alcohol test meal showed total acidity of 89° and 71° free hydrochloric acid. There was no gross blood in the stools initially, but later intermittent frankly tarry stools were noted. The Coombs test was negative; prothrombin time was 100 per cent of normal; the bleeding time 1.5 minutes; the coagulation time 8 minutes (Lee & White), and platelet count 211,200. The red blood fragility test showed beginning hemolysis at 0.44 per cent NaCl, and complete hemolysis at 0.28 per cent NaCl; the reticulocyte count was 0.5 per cent. Proctoscopic, esophagoscopic, and gastroscopic examinations, and roentgenologic studies of the gastrointestinal tract did not show any evidence of disease. A re-examination of the bone marrow on May 27, 1954 was reported as "Essentially normal marrow with acute changes in erythrocytes suggesting hypochromic anemia".

The hospital course was marked by continued loss of blood in the stools as shown by the frequent passage of tarry stools that gave consistent reactions for occult blood. It was necessary to transfuse the patient frequently in order to combat an increasing anemia. From May 7 through May 12 he received 3000 cc. of blood resulting in the following values: Red blood cells 4,290,000 per cu. mm., hemoglobin 12.9 Gm. per 100 cc., and hematocrit 43 per cent. During the next two weeks he did not receive blood transfusions and the laboratory values dropped to 3,750,000 red blood cells per cu. mm., hemoglobin 9.6 Gm. per 100 cc., and hematocrit 34 per cent.

A diagnosis of anemia due to blood loss from an ulcerated lesion of the gastrointestinal tract was made. Since no peptic ulceration of the esophagus, stomach or duodenum could be demonstrated and since there was no response to an ulcer regimen, surgical exploration was advised. A hemangioma was considered to be a likely cause of the bleeding.

Daily transfusions of 500 cc. were given from June 17 through June 21, 1954. A total of 17 transfusions of 500 cc. of blood were given before operation which raised the hemoglobin to 14.9 Gm. per 100 cc., the hematocrit to 47 per cent, and the red cell count to 5,570,000 per cu. mm.

The abdomen was explored. Following were the significant findings: 1. Obvious blood in the large bowel and distal ⅔ of the small bowel. 2. A small intussusception, 2 to 3 cm. in length, at about the junction of the jejunum and ileum. At this point, and obviously responsible for the intussusception, was a vascular tumor of the small bowel. Numerous fairly large, blue, tortuous venous channels could be seen through the serosa. By palpation a soft sessile tumor nodule measuring 2.5 cm. in diameter and 2 cm. in thickness protruding into the lumen of the bowel was demonstrated. 3. At a point approximately 24 inches proximal to the intussusception a second smaller angiomatous collection of vessels appearing much like an ecchymosis of the skin was found. This was about 1 cm. in diameter.

The smaller lesion was removed by excising it with a margin of normal intestinal wall, and closing the defect transversely. The larger tumor was removed by resecting a 7 cm. length segment of bowel and re-establishing continuity by an end to end anastomosis. The appendix was removed prophylactically.

Pathologic report of the larger lesion was as follows: Gross description: "The specimen

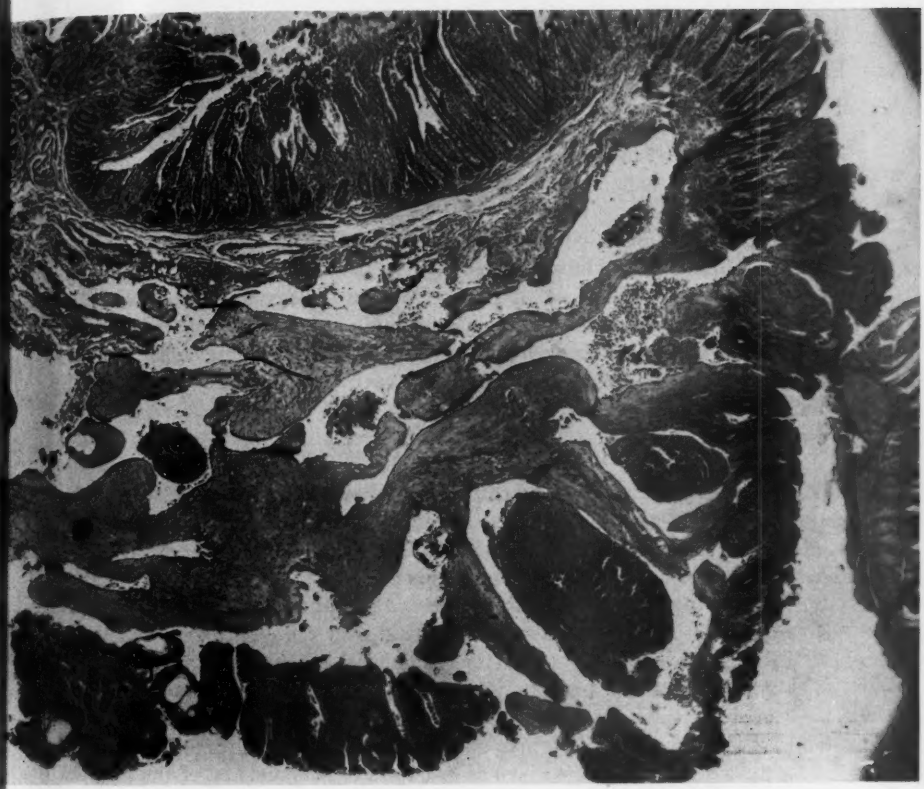

FIG. 1. Section through jejunal tumor showing large endothelal spaces within the lamina propria. Occasional recent thrombi are seen.

consists of a portion of small intestine which measures 5 cm. in length and when opened is 2 cm. in width. In the central portion on the mucosal aspect there is a slightly elevated area forming a polypoid tumor which in the fixed state shows a slight pale-gray discoloration. Cut surfaces show dilated vessels which appear to be filled with clotted blood."

Microscopic examination: "The covering mucosa shows an occasional focal area of increased friability and ulceration. In the submucosal tissues there are large dilated, endothelial lined spaces which are filled with blood and which are surrounded by hyalinized fibrous connective tissue. There are focal areas of recent hemorrhage within the lamina propria on either side of the lesion. There is no evidence of malignancy." Diagnosis: Cavernous hemangioma of jejunum with focal ulceration and recent interstitial hemorrhage (fig. 1).

The postoperative course was entirely uneventful. There has been no further evidence of anemia or blood loss. The patient has returned to gainful employment.

INCIDENCE

Hansen,[2] in a comprehensive review of hemangioma of the small intestine in 1941, stated that in 51,261 admissions to the surgical services of the hospital of

the University of Pennsylvania from 1922 to 1940, no case of hemangioma of the small intestine was found. He quoted statistics given by Raiford[7] and Merchant.[5] Raiford had found only 3 hemangiomas of the small intestine in 11,500 autopsies and 45,000 surgical specimens. Merchant found 3 hemangiomas of the small intestine in 7,340 autopsies and 50,775 surgical specimens. Gentry and associates[1] reported the incidence of all types of hemangioma of the entire gastrointestinal tract, benign and malignant, at the Mayo Clinic until July 1945 as 106 cases in 1,400,000 admissions, or roughly 1 case in 14,000 admissions. Fifty-one of the Mayo Clinic cases were hemangiomas of the small bowel; 46 of these gave no known symptoms during life and most of them were incidental autopsy findings. A few were incidental findings at laparotomy for other conditions. Of the cases seen at operation, 5 patients had their lesions resected; 1 of these was found incidentally in a specimen resected because of intestinal obstruction. Three of the 5 were reported well for over a five year period. Two patients had lesions too extensive to resect.

CLASSIFICATION AND PATHOLOGY

Pathologically, hemangiomas vary in type and distribution. Packard[6] has stated that the classification of hemangioma of the intestinal tract given by Kaijser[4] is a practical one. With this we are in agreement. Kaijser's classification divides hemangiomas into four groups as follows:

1. Multiple phlebectasis consists of dark bluish-red nodules ranging in size from pinhead to pea which are scattered over a limited portion of the intestine. These usually are in the submucosa although they may be subserous and usually are opposite the mesentery. Each one is connected with one of the smaller veins and is made up of communicating cavities. These probably are not true tumors but may be the result of some back pressure although they often are given as examples of hemangioma of the intestinal tract. There usually are no clinical symptoms and very little detectable hemorrhage in the canal. He listed 27 cases.
2. Cavernous hemangioma is divided into those developing by diffuse permeation in the intestinal wall and into those growing more compactly, thus frequently becoming polypoid. These are the angiomas particularly prone to cause hemorrhage or obstruction. He listed 32 cases.
3. Simple hemangioma or capillary hemangioma is a network of dilated capillaries as compared to the large blood filled, thinly lined communicating cavities of the cavernosa type. The simple type usually appears as a round submucous tumor, up to a plum in size, protruding into the canal and covered by normal mucosa. Kaijser found 7 cases.
4. Angiomatosis is his heading to cover the occurrence of multiple angiomas varying in structure from the usual cavernous type to one rich in cells and stroma, some even suggesting angiosarcoma. Those in the intestine may be tiny nodules or plaques of considerable size but do not infiltrate and usually are on the surface away from the mesentery. Seven cases were reported.

Gentry and associates have proposed a much more comprehensive, but cumbersome, classification that has the virtue of providing a separate category for malignant tumors. They reviewed the world literature through June 1945 reporting a total of 283 benign and 61 malignant vascular malformations and tumors of the gastrointestinal tract, including 94 benign and 16 malignant cases from the Mayo Clinic.

CLINICAL MANIFESTATIONS

The history of intestinal bleeding with secondary anemia is the most prominent complaint. Enlargement of the spleen and liver usually is absent. Abdominal pain, vomiting, tenderness, and disturbed bowel sounds usually are absent, but may be present if the lesions cause obstruction. There usually will be the history of frank hemorrhage in the stools or tarry stools and weakness.

On physical examination findings other than those due to the secondary anemia are infrequent. Sometimes hemangiomas will be found in the oral cavity or rectum. If these or skin hemangiomas are found, one should suspect the presence of an intestinal hemangioma as the cause of bleeding.

The diagnosis of hemangioma usually is made at laparotomy. It can be suspected only if one is aware that it may exist. Then history and physical findings usually will show only evidence of intestinal bleeding. Roentgenologic studies of the upper gastrointestinal tract usually shows nothing abnormal. It probably would be possible occasionally to demonstrate these lesions with a careful roentgenologic study of the small intestine, but such examinations very often are overlooked.

TREATMENT

The treatment of hemangioma of the gastrointestinal tract is blood replacement, correction of chemical imbalance, and resection of the lesion or lesions when technically feasible. Palliative forms of treatment such as irradiation, cauterization and injection of sclerosing solutions should be used only if resection is impossible. Extensive or multiple resections may at times be considered. For example Heycock and Dickinson[3] reported a case of multiple hemangiomas of the large and small bowel in a child aged 6 that had resulted in multiple serious gastrointestinal hemorrhages. During the course of two laparotomies 30 separate hemangiomas were excised with no recurrence of bleeding up to the time of the report.

Laparotomy usually is an indicated procedure. Careful exploration by palpation, visualization and transillumination of the bowel with a good light in a darkened room will reveal most of the lesions.

SUMMARY

A case report of polypoid cavernous hemangioma of the jejunum is presented. A classification of vascular tumors of the gastrointestinal tract as suggested by Kaijser[4] again is brought to your attention.

A brief discussion of the clinical aspects of the disease is presented.

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SPONTANEOUS RUPTURE OF THE STOMACH

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In the past decade only 2 cases of spontaneous rupture of the stomach, without associated trauma or pre-existing pathologic lesions, have been reported in the American medical literature. One of these, reported by Pendergrass and Booth,[3] was of a 3 day old baby who had two perforations in an otherwise normal stomach. The other case, reported by R. F. Peterson,[4] was of a young woman who died following extraction of teeth under general anesthesia. The autopsy revealed a ruptured stomach.

Glassman[1] reported 14 cases of spontaneous rupture of the stomach in 1929, and in 1936 Wolf[5] reviewed the literature and listed 17 cases, some from Glassman's series. Lemmon and Paschall[2] added 14 cases in 1941, bringing the total to 31 cases. In this group only 5 patients were operated upon, 2 of whom recovered. Many of the patients in this series were from foreign countries. Since Lemmon and Paschall reviewed the literature in 1941, there have been 15 additional cases reported in the foreign literature. Assuming these 15 cases to be true spontaneous ruptures, the total number of cases, including the case to be reported, is 49. In none of these patients was a diagnosis made prior to operation or autopsy.

Clinically, the patients with acute rupture of the stomach present a picture of severe shock and prostration, usually with marked pain. The distention of the abdomen, with increase in gastric pressure is a striking feature. The distention is generalized and may be due to the pneumoperitoneum, which usually is present. The abdomen is tympanitic, has a board like rigidity, with marked generalized tenderness, and absence of peristaltic sounds. In most patients, death occurs within a few hours after the onset of the illness.

This condition occurs in all ages, but the incidence is highest in the middle aged group. The rupture of the gastric wall usually occurs on the lesser curvature, and at autopsy or operation the abdomen is found greatly distended, and the peritoneal cavity is grossly contaminated with undigested and partially digested foods and liquids. The distention usually is associated with overloading or overeating, and sometimes is combined with the administration of antacids, such as bicarbonate of soda.

CASE REPORT

The case here reported is of an 85 year old white woman who felt well until shortly after eating a large Christmas dinner, at which time she experienced a sensation of fullness in her abdomen. This was associated with a desire to belch or regurgitate, attempts at which were unsuccessful. She took a tablet containing an antacid. She had been taking antacid tablets

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for years for similar attacks of abdominal discomfort. However, she continued to have increasing abdominal discomfort following taking the tablets. The pain in the abdomen was generalized and very severe. When she was first seen, about two hours after eating the meal, she was very restless, changing positions frequently in the bed, and occasionally crying out because of the extreme pain. She was rational, but due to the great discomfort, was unable to answer questions in detail. The family gave the information that she generally was in fairly good health, was an active woman for her age, had a good appetite and frequently was inclined to over eat. She had known gallbladder disease, which caused infrequent bouts of abdominal discomfort, but usually was relieved by taking some tablets.

Examination revealed an acutely ill, well developed, elderly, white woman who appeared to be in acute distress. The blood pressure was 150/115. The pulse was 104 per minute and regular. There was visible distention of the abdomen, with a board like rigidity throughout. The abdomen was tympanitic to percussion and there was generalized tenderness. Peristaltic sounds were absent. A Levine tube was inserted but nothing was obtained except a small quantity of mucus. The tube was withdrawn and reinserted but no gastric contents were obtained. She was sent to the hospital via ambulance, and just before reaching the hospital, vomited a large quantity of undigested food and brownish liquid material. Shortly after reaching the emergency room, she aspirated a small quantity of vomitus and became cyanotic. Tracheal suction immediately was instituted, but the cyanosis progressed, the blood pressure dropped, and she died a few minutes after being admitted to the emergency room.

Autopsy showed a well developed, well nourished, 85 year old white female. Particles of undigested food were protruding from the mouth. The abdomen was protuberant, and on opening the abdomen a considerable quantity of gas escaped under pressure. The abdomen was partially filled by partially digested food. This material was escaping from a rent in the stomach, which was located on the lesser curvature, approximately 10 cm. from the esophagogastric junction. The rent measured 6 cm. in diameter. There was approximately 300 cc. of thin yellowish, fetid smelling fluid in the abdominal cavity, as well as particles of partially digested food. Loops of small and large bowel were dilated. The intestines were examined carefully, but there was no evidence of extrinsic or intrinsic obstruction. The stomach was dilated and filled with partially digested food. There was no evidence of ulcer or any extrinsic or intrinsic lesions. Examination of the chest showed a moderate degree of postmortem congestion in the lungs, and on opening the trachea a moderate quantity of partially digested food was found in the trachea and in both the right and left lungs.

Incidental findings were chronic cholecystitis with cholelithiasis, a cholecystoduodenal fistula, which was asymptomatic at the time, marked generalized atherosclerosis, and severe arteriolar-nephrosclerosis.

Dr. H. T. Hayes of Baylor Hospital did the autopsy. His conclusions were that the aspiration of food particles in the lung probably was the primary cause of death, and that the shock associated with the acute dilatation and subsequent rupture of the stomach, with the formation of chemical peritonitis was a contributing cause in this patient's death.

CONCLUSIONS

The cause of rupture of the stomach in this patient was assumed to be due to overeating, plus the ingestion of antacids, producing marked gaseous distention. According to Glassman's[1] theory, distention paralyzes the nervous mechanism, so that the overfilled stomach does not empty, and with subsequent fermentation and further dilatation, the stomach eventually ruptures. This mechanism, probably associated with cardiospasm and pylorospasm, is thought to be the precipitating factor in this case.

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GALLSTONE ILEUS PRODUCED BY A STONE PASSED THROUGH THE AMPULLA OF VATER*

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Intestinal obstruction due to gallstone is becoming less frequent because of the more widely practiced removal of all gallbladders containing stones. The stone usually enters the intestinal tract through a fistula between the gallbladder and the bowel. The following case illustrates the more unusual passage of the stone through the common bile duct and the ampulla of Vater.

CASE REPORT

The patient was an 87 year old woman. She was admitted first to The New York Hospital on Oct. 14, 1952 because of a steadily enlarging mass over the radial aspect of the left proximal forearm. This mass had appeared six months previously and caused only mild, aching discomfort.

The *past history* included a fracture of the right hip three years previously with resulting nonunion. A cecostomy had been performed two and a half years ago at another hospital because of diverticulitis. The appendix was removed. She denied all gastrointestinal symptoms suggestive of gallbladder disease. There had been progressive loss of vision for several years.

Physical examination showed a well preserved, elderly woman. There was marked diminution of vision with bilateral senile cataracts. There was moderate pulmonary emphysema. The cardiac rhythm was irregular; the blood pressure was 140/100. The abdomen was negative except for well healed right lower quadrant incision. Marked limitation of motion of the right hip was present. Over the radial aspect of the left proximal forearm there was a firm, nontender mass 17 by 10 cm. in size fixed to the deep tissues but not to the skin or bone.

Laboratory: Hemoglobin 13.5 Gm.; hematocrit 36; blood urea nitrogen 13 mg. per cent; serum protein 5.6 Gm. with A/G ratio of 3.4/2.2; alkaline phosphatase 4.8; prothrombin time normal; elecrocardiograph showed auricular fibrillation with left axis deviation.

Roentgenologic examinations: Chest film showed evidence of metastatic tumor in the left lower lung field; spine films revealed evidence of bone destruction of fifth lumbar and first sacral vertebrae; right hip showed a nonunited fracture of femoral neck; right forearm film showed a large, soft tissue mass without bone involvement.

Course: On Oct. 22, 1952 the tumor mass of the left forearm was excised. Pathologic examination showed a rhabdomyosarcoma. The postoperative course was uneventful. Because of a recurrent mass in the region of this excision, the patient was readmitted and on May 6, 1953, the recurrent tumor (5 by 4 cm.) was excised under local anesthesia. One week later, on May 13, a right cataract extraction was done. Following the latter operation, the patient developed minimal generalized abdominal discomfort which persisted during the following nine days. She vomited several times and a gastric tube was inserted and placed on suction. Abdominal examinations showed persistent moderate abdominal distention with mild left lower quadrant tenderness. Repeat roentgenograms of the abdomen showed distended large and small bowel. A barium enema four days after the onset of symptoms showed diverticulitis of the sigmoid with marked spasm. The patient remained afebrile. The white blood cell count was elevated to 13,700 per cu. mm. with slight shift to the left.

Because of the persistence of symptoms and the evidence of diverticulitis, a transverse

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colostomy was done on May 22 under local anesthesia. The colon was moderately distended. The abdomen was not explored.

There was slow decrease in the abdominal distention after the colostomy and the patient was taking a soft diet by the ninth postoperative day. At this time the abdomen again became distended and the patient had crampy abdominal pain. Colostomy drainage stopped. Gastric suction was reinstituted. Abdominal roentgenograms suggested small bowel obstruction. Subsequent review of these films showed air in the biliary tract (figs. 1, 2).

On June 5, 16 days after the colostomy, the abdomen was explored under general anesthesia. A large gallstone (3.5 by 3 by 3 cm.) was found obstructing the distal ileum about 6

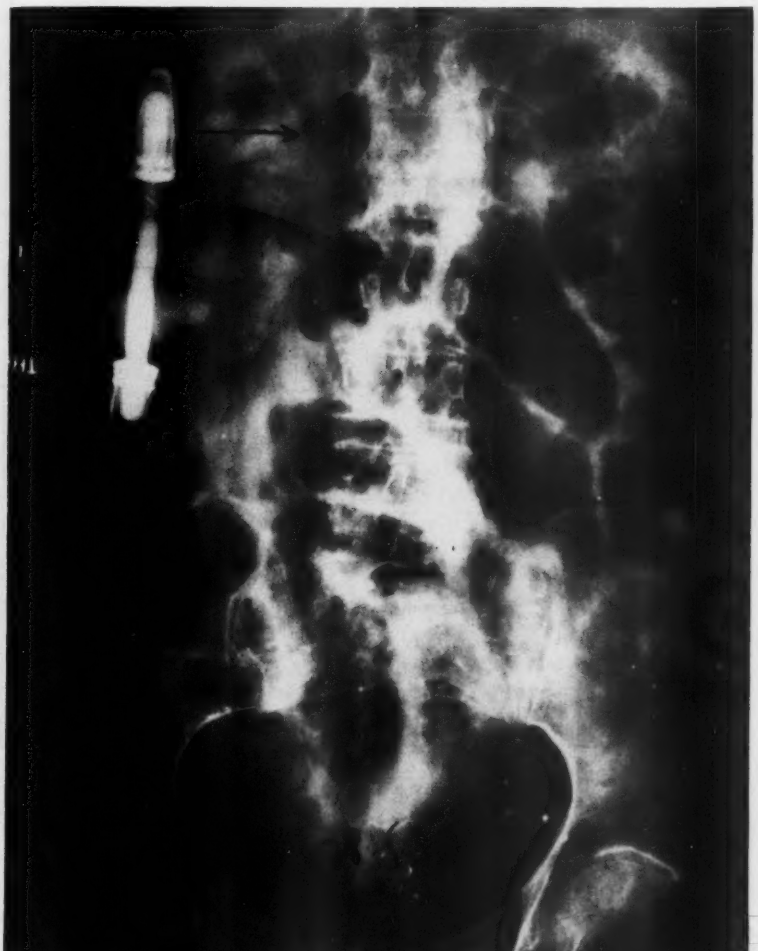

FIG. 1. Roentgenogram of abdomen taken before removal of the gallstone from the ileum. Note the gas in the bile duct.

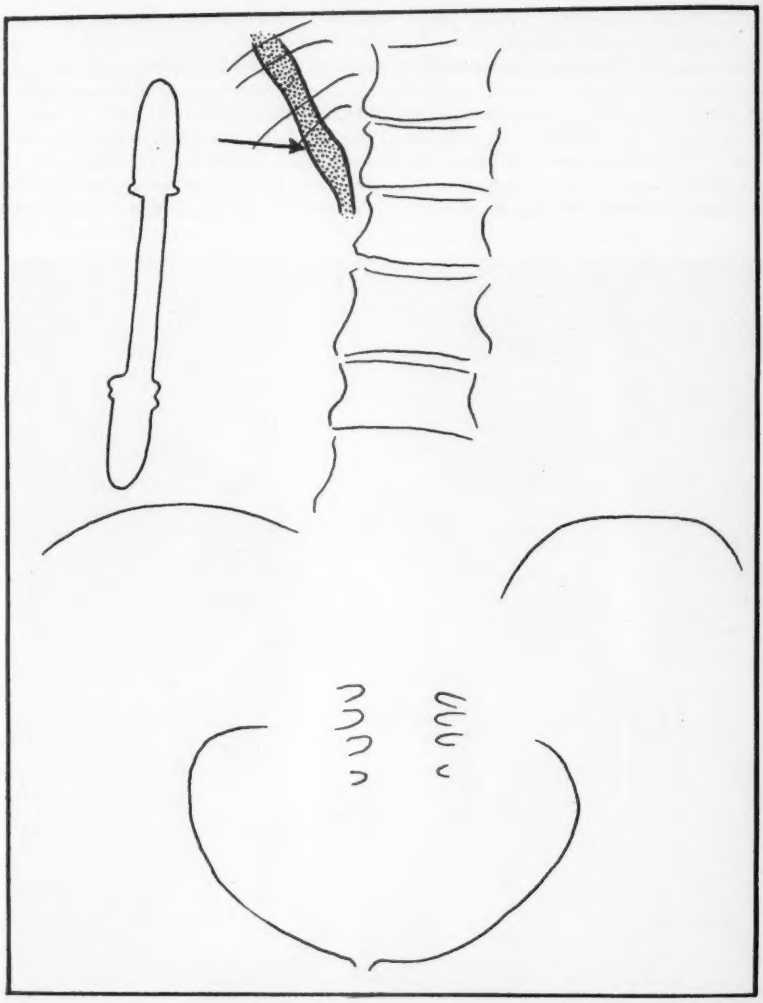

FIG. 2. Sketch showing dilated common bile duct

inches proximal to the ileocecal valve. The intestine was of questionable viability at this point and for this reason was resected and end to end anastomosis was made.

On the fourth postoperative day fecal drainage appeared in the incision. Thereafter, an extensive cellulitis of the abdominal wall developed. The postoperative course was further complicated by a coronary occlusion (fifth postoperative day) and a cerebral hemorrhage (eighth postoperative day). The patient's condition deteriorated slowly and she died on July 17, 1953, five and a half weeks after the last operation.

Autopsy showed a loculated peritonitis with a fecal fistula at the site of the disrupted anastomosis. The gallbladder was thickened and sclerotic. It contained no stones. There

was no evidence of fistula between the gallbladder and the intestinal tract. The common duct was much dilated and entered the duodenum through a patulous ampullary opening. There was metastatic rhabdomyosarcoma in the left lung. Multiple diverticula were present in the sigmoid colon.

COMMENT

Gallstones large enough to obstruct the bowel almost always enter the bowel through an internal biliary fistula.[2, 5] In the majority of patients the fistula is between the gallbladder and the duodenum but it may be between the common bile duct and the duodenum or between the gallbladder and the ileum, jejunum, colon, or stomach.

Doubt has been expressed by some authors[3, 7] that a gallstone of sufficient size to obstruct the bowel can pass through the ampulla. However, a small number of cases are reported in the literature which indicates that such a sequence of events is possible.

Courvoisier,[1] in 1890, stated that in 7 of 35 cases of intestinal obstruction by gallstones, a fistulous connection between the intestinal and biliary tracts could not be demonstrated. This is most unusual, for only 2 other cases are found in the literature which exhibit a similar situation.

Murphy,[4] in 1910, described a stone 4 inches in diameter in the small bowel. There was no biliary intestinal fistula described and the common duct was dilated. The size of the duct was said to be the size of the index finger and the author thought that it had shrunk down to that size after passage of the stone. These findings were at autopsy. A detailed description is not given.

Snyder,[8] in 1938, reported a case with the autopsy finding of a stone the size of a large walnut near the junction of the jejunum and ileum. The gallbladder contained no stone and there was no fistula demonstrable between the biliary and intestinal tracts. The cystic duct, common duct, and ampulla of Vater were markedly dilated permitting easy passage of one index finger from the gallbladder to the duodenum.

Scofield,[6] in 1930, described a patient in whom the duodenum contained "10 moderately sized gallstones." There was no fistula between the gallbladder and duodenum. No statement was made regarding a choledochoduodenal fistula and apparently none was demonstrable. However, this patient did not have intestinal obstruction and therefore is not comparable to the cases described above.

SUMMARY

A case of intestinal obstruction due to a gallstone is presented in which the stone presumably entered the intestinal tract through the common bile duct and the ampulla of Vater. No biliary-intestinal fistula was found. The common duct and papilla of Vater were dilated. This represents a very infrequent occurrence as evidenced by the small number of such cases described in the literature.

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BOOK REVIEWS

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

Vaginal Hysterectomy: Indications, Technique and Complications, by LAMAN A. GRAY, A.B., M.D., F.A.C.S., Associate Professor, Obstetrics and Gynecology, University of Louisville School of Medicine, Louisville, Kentucky; Area Consultant Gynecologist, Veterans Administration; Consultant Gynecologist, Nichols Veterans Administration Hospital; Active Staff, Norton Memorial Infirmary and Louisville General Hospital. Southern Surgical Association, Central Association of Obstetricians and Gynecologists; American Urological Association (Southeastern Section); American Academy of Obstetrics and Gynecology; Society of Pelvic Surgeons. Charles C Thomas, Springfield, Illinois. Price $4.75.

The purpose of this monograph is to instruct the occasional hysterectomist in the judicious use of vaginal hysterectomy. After a brief but complete resume of the literature, there is a very important chapter on indications. Being a "middle-of-the-roader" as far as operative approach is concerned, Doctor Gray attempts to delineate indications for the vaginal and abdominal procedures. The attempt is noble but clarification of indications for vaginal surgery is more apparent than real since the author lumps various and sundry conditions under his "combined syndrome".

The chapter on technic is good in that it is detailed and specific. One wishes, perhaps, that the correlation between text and illustrations were a little bit closer but the latter in general are excellent. Postoperative complications are explored and fully dealt with. The text is for the most part lucid. Aside from Doctor Gray's helpful hints on technic, there is not much new in "Vaginal Hysterectomy". However, the book is timely, covers its subject well, and should fulfill its purpose in being of value to the occasional hysterectomist.

EUGENE W. J. PEARCE, M.D.

Textbook of Operative Surgery by ERIC L. FARQUHARSON, M.D., F.R.C.S.Ed., F.R.C.S.Eng.; Surgeon, Royal Infirmary of Edinburgh; Member of Clinical Teaching Staff, University of Edinburgh. Visiting Surgeon, Berwick Infirmary, Berwick-on-Tweed; Visiting Surgeon, General Hospital, Kirkcaldy. Examiner, Royal College of Surgeons of England. E. & S. Livingstone, Ltd., Edinburgh and London. Price $15.00.

In the preface the author has stated that he has endeavored to describe, in detail and with adequate illustrations, all of the operations which the junior in general surgery is likely to undertake himself, and also the more common operations in which he may be called upon to assist. He includes a description of operations in specialized fields of surgery which the general surgeon may be required to undertake when more expert help is not available. A few of the gynecologic operations might well have been added. This plan of an operative surgery is laudable and is placing the emphasis where it is most needed.

There are 853 pages and 623 numbered illustrations in this book. The format and the quality of paper and printing are good. The illustrations are excellent and add immeasurably to the quality and usefulness of the work. The contents are listed, with a few exceptions, as operations upon the various anatomic structures of the body. A description of the anatomy is included where such information is important.

Surgeons are prone to disagree concerning points in technic which may be considered by some as of minor importance. For example, I would not recommend the type of incision used by the author for radical mastectomy, or any other incision for this operation which crosses the axilla and predisposes to scar formation between the arm and chest wall. Also some surgeons would not agree that the author's method of closure of a duodenal perforation is the best, but prefer to close the opening in the duodenum with an omental tab to avoid constriction.

This is an excellent book. It is an admirable presentation of the subject which will be a useful guide for the young surgeon. Some of the more experienced surgeons would profit by its review.

THOMAS G. ORR, M.D.

Cardiac Anomalies: A Clinicopathologic Correlation, by VINCENT MORAGUES, M.D., Associate Professor of Pathology, Creighton University School of Medicine; Associate Pathologist, Creighton Memorial St. Joseph's Hospital, Omaha, Nebraska; Formerly Associate Professor of Pathology, St. Louis University School of Medicine, St. Louis, and CHESTER P. LYNXWILER, M.D., Assistant Professor of Pediatrics, St. Louis University School of Medicine, St. Louis; Director, Pediatric Heart Clinic, Firmin Desloge Hospital, St. Louis. The Williams & Wilkins Company, Baltimore, 1954. Price $6.50.

This book is a small picture album illustrating the majority of congenital cardiac lesions. Surprisingly, aberrant pulmonary veins and anomalous coronary arteries are not included. Roentgenograms, cardiograms and gross specimens are illustrated. Frequent lung biopsies also are shown.

Physiologic data from right heart catheterization is lacking. The narration is fragmentary. Few angiocardiograms are shown. The photographs of gross specimens occasionally are blurred.

No gross errors were noted. The book is attractively printed and bound. The primary value of the book would be for those seeking elementary information about congenital heart disease.

E. GREY DIMOND, M.D.

Surgery of the Caecum and Colon by STANLEY AYLETT, M.B.E., M.B., B.S., B.Sc., F.R.C.S. Surgeon, The Westminster Hospital Teaching Group (Gordon Hospital), The Metropolitan Hospital, Potters Bar and District Hospital; Teacher in Surgery, The University of London. E. & S. Livingstone, Ltd., Edinburgh and London, 1954. The Williams & Wilkins Co., Baltimore. Price $9.00.

This monograph of 295 pages with 142 illustrations thoroughly covers the anatomy, physiology, pathology and clinical aspects of the caecum and colon and the diseases involving this segment of the gastrointestinal tract. A sharp division between the colon and the rectum seems artificial. The author apparently has abandoned that area beyond the peritoneal reflection to the "specialist", carefully pointing out that "surgery of the caecum and colon is rightly within the province of the general surgeon".

The section on anatomy, physiology and pathology adequately covers the standard material. The chapter on choice of operation for cancer is especially well presented and illustrated. Preoperative and postoperative management, as well as operative technic, are presented in great detail as those employed by the author and the surgical staff of the Gordon Hospital in their day to day handling of these cases. The reader will find, therefore, many small differences between the practices outlined here and those in common usage in this country.

The material is well organized and well presented. Those wishing to read further on the various topics presented will find a well selected list of references at the end of each chapter.

GEORGE A. HIGGINS, M.D.

The Kidneys. Ciba Foundation Symposium, edited by A. A. G. LEWIS, B.Sc., M.D., M.R.C.P. and G.E.W. WOLSTENHOLME, O.B.E., M.A., M.B., B.Ch. Little Brown & Company, Boston, 1954, 333 pages, numerous illustrations and charts. Price: $6.00.

The previous Ciba Symposia have all been excellent and this new Symposium on the kidney, held and recorded in July, 1953, upholds the high standard of the previous publications. All of the papers presented at this Symposium are excellent and bring to us in a compact form the latest information regarding the physiology and pathology of the kidney. Each paper has stimulated a good deal of discussion and the comments of the discussors are

often more valuable than the paper itself. The contributions by the American physiologists, Dr. Jean Oliver, Dr. J. P. Merrill, Dr. R. F. Pitts and Dr. J. V. Taggart are outstanding and the obviously good international spirit of all the participants is evident in their discussions. This volume is recommended to all who have more than a casual interest in the physiology of the kidney.

WILLIAM L. VALK, M.D.

BOOKS RECEIVED

Books received are acknowledged in this section, and such acknowledgement must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

A Method of Balanced Anesthesia in General Surgery, Obstetrics and Dentistry, by SYLVAN M. SHANE, D.D.S., Attending Anesthesiologist, Lutheran Hospital of Maryland; Chief of Anesthesiology, Doctors Hospital of Baltimore; Consultant in Anesthesiology, Spring Grove Hospital of Maryland, and HARRY ASHMAN, M.D., Chief of Anesthesiology, Lutheran Hospital of Maryland. Lowry & Volz, Baltimore, 1955. Price $2.25.

Surgery of the Elbow, by FREDERICK M. SMITH, M.D., Associate Professor of Clinical Orthopedic Surgery, Columbia University, New York City. Charles C Thomas, Publisher, Springfield, Illinois, U. S. A. Price $10.00.

Clinical Neurosurgery, Volume I: Proceedings of the Congress of Neurological Surgeons, New Orleans, Louisiana. Baltimore, The Williams & Wilkins Company, 1955. Price $8.00.